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Xpey' Relational Environments: an analytic framework for conceptualizing Indigenous health equity

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Abstract

Introduction: Both health equity research and Indigenous health research are driven by the goal of promoting equitable health outcomes among marginalized and underserved populations. However, the two fields often operate independently, without collaboration. As a result, Indigenous populations are underrepresented in health equity research relative to the disproportionate burden of health inequities they experience. In this methodological article, we present Xpey' Relational Environments, an analytic framework that maps some of the barriers and facilitators to health equity for Indigenous peoples.

Methods: Health equity research needs to include a focus on Indigenous populations and Indigenized methodologies, a shift that could fill gaps in knowledge with the potential to contribute to 'closing the gap' in Indigenous health. With this in mind, the Equity Lens in Public Health (ELPH) research program adopted the Xpey' Relational Environments framework to add a focus on Indigenous populations to our research on the prioritization and implementation of health equity. The analytic framework introduced an Indigenized health equity lens to our methodology, which facilitated the identification of social, structural and systemic determinants of Indigenous health. To test the framework, we conducted a pilot case study of one of British Columbia's regional health authorities, which included a review of core policies and plans as well as interviews and focus groups with frontline staff, managers and senior executives.

Conclusion: ELPH's application of Xpey' Relational Environments serves as an example of the analytic framework's utility for exploring and conceptualizing Indigenous health equity in BC's public health system. Future applications of the framework should be embedded in Indigenous research methodologies.

Keywords: *health equity, health services accessibility, public health, research methodology, Indigenous populations*

Highlights

- Indigenous peoples in Canada face inequities in access to health services as well as health outcomes, which are unnecessary and unjust.
- There are various determinants of Indigenous health, including protective features that promote well-being and resilience, as well as risk factors that can produce unfavorable circumstances or hinder health.
- Indigenous health equity is a critical issue in BC's public health system and needs to be a priority for researchers, policy makers and practitioners.
- Making Indigenous populations a priority in public health policy, practice and research can contribute to improving the overall health and wellness of Indigenous peoples.

Introduction

Within Canada and abroad, two emerging branches of health research are rapidly advancing and have the potential to inform each other: health equity research and Indigenous health research. Health equity research is the investigation of disparities in health status or the delivery of health care;[1] it is also the study of strategies, programs or policies to reduce/ eliminate inequities and promote health equity. Indigenous health research is the study of the health and well-being of Indigenous populations, which often entails the application of Indigenized or decolonizing research methods that infuse Indigenous ways of knowing and cultural protocols into research practice.[2,3]

Both branches of research are driven by the goal of promoting equitable health outcomes among marginalized and underserved populations; however, the two fields tend to operate independently, often without collaboration.[2] This disconnect may have an impact on how Indigenous health equity is investigated, if at all.[1] Health equity research needs to include a focus on Indigenous populations and Indigenized methodologies. This shift could fill this gap in knowledge with the potential to contribute to 'closing the gap' in Indigenous health.

In this article, we present Xpey' Relational Environments, an analytic framework designed for conceptualizing the physical, interpersonal and institutional settings where Indigenous health equity may or may not be manifest. We showcase the framework's application within the Equity Lens in Public Health (ELPH) research

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program as an example of its relevance to analyzing Indigenous health equity within public health environments. Xpey' Relational Environments has been used by the ELPH research team to explore strategies used by British Columbia's (BC's) public health system to reduce inequities and enhance health equity for Indigenous peoples, including First Nations, Inuit and Métis peoples. The specific research questions that ELPH seeks to answer are: (1) What are the barriers and facilitators to health equity within BC's public health system? (2) What specific strategies are proposed and implemented by health authorities to address health equity? (3) To what extent has health equity been identified and prioritized within health authorities as reflected in core documents and plans? and (4) What are the contextual influences on priority setting and equity goals at the organizational and broader systems levels? Xpey' Relational Environments adds a specific focus on Indigenous peoples in answer to these questions and an Indigenous lens to the analysis of the data.

Locating ourselves in this research

Alexandra Kent

I locate myself in this research as a fifth-generation Canadian settler of British and Dutch ancestry. Though I do not care to label myself as such, I am positioned in society as a white, Western-educated, English-speaking, able-bodied, heterosexual woman. I acknowledge that by embodying this intersectional identity, I am privileged as a member of several dominant groups. Furthermore, as a settler living on the unceded territories of Songhees, Esquimalt and W̱SÁNEĆ peoples, I am implicated in Canada's history of colonialism as a beneficiary of the dispossession and subjugation of Indigenous peoples. I am approaching this research with humility and the understanding that I do not represent Indigenous peoples or their knowledge, worldviews, and cultural lenses. I hope to contribute to the decolonization agenda as a non-Indigenous ally through critical engagement with public health research. I have had the privilege of working as a Research Fellow, and more recently a Research Assistant, with the Equity Lens in Public Health (ELPH) research program and the Centre for Indigenous Research and Community-Led Engagement (CIRCLE), who partnered to add an Indigenous focus and Indigenous knowledge to the ELPH project. Working with both ELPH and CIRCLE has given me a unique perspective that integrates Western academia with Indigenous ways of knowing, much like Two-Eyed Seeing.[4] It is from this position and with critical awareness of my location that I present Xpey' Relational Environments with my colleagues.

Charlotte Loppie

I am of Mi'kmaq and French Acadian ancestry from Nova Scotia. I have been a grateful visitor in the territory of the Songhees, Esquimalt and W̱SÁNEĆ peoples since 2009. I have served the health research needs of Indigenous communities and collectivities since 1995; I teach Indigenous health courses in the School of Public Health and Social Policy at the University of Victoria, where I also serve as the Director of the Centre for Indigenous Research and Community-Led Engagement (CIRCLE). I am not an Investigator on the ELPH project but was asked by the team to co-develop the Xpey' Relational Environments framework, which I hope will support Indigenous health equity in BC and elsewhere.

Jeannine Carriere

My Cree name is Sohki Aski Esquao, which means Strong Earth Woman. My English name is Jeannine Carriere. I am a Métis woman who comes from the Red River area of Manitoba and I have been living in the territory of the Cowichan peoples on Vancouver Island since 2005. I have been teaching since 1994 and at the University of Victoria on the territory of the Songhees, Esquimalt and W̱SÁNEĆ peoples in the School of Social Work since 2005. My areas of research include adoption and identity for Indigenous children, Indigenous research methodologies, Metis child and family wellbeing and family relationships in sex work. Through these areas of interest I have been fortunate to publish and develop networks with some amazing folks such as those affiliated with the ELPH project. For this, I am grateful.

Marjorie MacDonald

I am positioned in this research as a well-educated, white, able-bodied, middle-class heterosexual woman. This has provided me with privileges not open to many others in society. Although my great-grandmother on my mother's side was Cree, her descendents were all raised as white settlers, sadly without knowledge about or acknowledgement of their Cree heritage. Thus, we are all implicated in the colonization and oppression of Indigenous peoples in this country, including our own relatives. I lived and worked on the Blackfoot Reserve at Gleichen Alberta and came to learn and appreciate the traditions and worldview of the Blackfoot. This experience led me to make a life-long commitment to public health with its communitarian values and collectivist ethic. Yet, I claim no special knowledge, position, or privilege in this research related to this experience. As a committed ally, I acknowledge, with gratitude and humility, my visitor status in the unceded territories of the Songhees, Esquimalt and W̱SÁNEĆ peoples.

Bernadette Pauly

I am a third generation settler of German ancestry. I am positioned in society as a white, able bodied, well-educated heterosexual middle class woman. I have had opportunities and privileges open to me as a result of my position in society. I am a settler living and working on the unceded territories of the Songhees, Esquimalt and W̱SÁNEĆ peoples since 2000. As such, I am implicated in the colonial history of Canada and actions of many Canadian settlers that resulted in the displacement and oppression of Canada's Indigenous peoples. In this research, I recognize that my position is not one of belonging to or intimate knowledge of Indigenous knowledge, worldviews, or cultural lenses. My aim is to approach this research respectfully and humbly in the hopes that critical engagement will contribute to the decolonization agenda. I am extremely grateful and appreciative of the opportunity to work with Indigenous scholars in this work and in other research, and have learnt an enormous amount through these processes. For that, I am thankful.

Equity Lens in Public Health research program

ELPH is a five-year program of research funded by CIHR in 2011 that grew out of the Core Public Health Functions Research Initiative (CPHFRI), which was initiated in 2005. Both CPHFRI and ELPH are specific to BC's public health system, and operate in close partnership with BC's five regional health authorities, the Ministry of Health, and the Provincial Health Services Authority (PHSA), among other partners. These partnerships were solidified prior to ELPH's inception in 2011. The First Nations

Health Authority (FNHA) was invited to partner with ELPH in 2013 (when it was formally established); we respect their decision to not participate and take this into consideration as a limitation of the study.

The ELPH research program is dedicated to the development and application of an equity lens in public health and the implications for reducing health inequities.[5] It has a particular focus on mental health promotion and preventing mental illness and the harms of substance use. The project is organized into four distinct but interrelated studies. Xpey' Relational Environments was used in a case study within ELPH Study 1: Health Equity Priorities and Strategies, the intent of which is to conduct individual case studies among BC's five regional health authorities to determine: (1) current activity on health equity and inequity reduction; (2) whether and how health equity issues have been prioritized; (3) the contextual influences on priorities and health equity plans/strategies; and (4) how and what explains these changes over the course of the study. Each health authority represents an individual case to capture contextual influences at the regional level and facilitate comparison across cases. The case study design also enables comparative analysis across times (baseline and follow-up) for assessment of changes in the uptake and implementation of health equity as a priority.

The ELPH project uses situational analysis, which is an approach to research using post-modern grounded theorizing methodology to identify and describe social worlds and arenas of action to understand the human and non-human elements, interactions and context within a specified situation.[6] Consistent with Clarke's[6] methodology, we are using situational analysis to open up the data and to facilitate analysis of multiple connections and relationships that can influence activities. Ordered, relational, social world, and positional maps are visual representations for understanding the phenomena of interest and the complexity inherent in a situation. As an outcome of the ELPH study, we will produce regional case reports and an overall provincial level analysis that summarizes findings related to application and implementation of health equity across health authorities.[5] The situational analysis will be reported elsewhere.

Background

In the original ELPH proposal, health inequities among Indigenous peoples, including First Nations, Inuit and Métis peoples, were highlighted as a concern in BC along with health inequities of other sub-populations. The ELPH team identified the need to pay special attention to health equity for Indigenous peoples, which would require a more culturally relevant analytic framework. Consequently, the Principal Investigators approached Indigenous researchers, Dr. Charlotte Loppie (CL, formerly Reading) and Dr. Jeannine Carriere (JC), who developed the Xpey' Relational Environments framework which serves as the basis of a parallel analysis that uses an Indigenous lens to explore the role of public health in Indigenous health equity.

The Haudenosaunee Guswenta model (or Two Row Wampum)

The ELPH team acknowledges the Haudenosaunee Guswenta, or Two Row Wampum, as a model for conceptualizing the relationship between ELPH's use of situational analysis methodology and the Xpey' Relational Environments approach. The Guswenta, or Two Row Wampum, is a beaded belt that was exchanged at the Treaty of Niagara in 1764. The belt depicts two boats (a First Nations canoe and a European ship) traveling side by side down a river, neither of them trying to steer the other's vessel or intersect the other's path, symbolizing mutual respect and non-interference.[7] Like the Two Row Wampum, our analysis represents parallel processes that share an overarching purpose and a common data set. Our research recognizes Indigenous and Western approaches as distinctly yet equally significant, and draws together the strengths of both to allow for a "wider, deeper, and more generative 'field of view' than might either of these perspectives [provide] in permanent isolation".[4] Furthermore, we take caution not to merge the two knowledge systems into one or try to force Indigenous knowledge into a Western paradigm.

State of knowledge

ELPH's research is grounded in a body of literature on health equity and public health systems. Although knowledge and awareness are expanding in these areas, relatively little research has bridged the two or further linked them to Indigenous health. In 2010, the Canadian Coalition for Global Health Research partnered with the Centre for Aboriginal Health Research [what is now CIRCLE] on a project titled, "Linking Equity Methods Research and Global Indigenous Health Research." The project included an environmental scan, tools inventory, and workshop to develop a work plan for forwarding an agenda for collaboration between the two fields of research.[7,8] The investigators concluded that, "While advancements in equity methods research have been made in the past decade... more work was needed on research evidence focused on the health of Indigenous populations".[8,p.2] The following sections will briefly outline some of the pertinent background information to set the context for our research.

Health equity

The standard definition of health inequity used in research circles is "differences in health which are not only unnecessary and avoidable but, in addition, are considered unfair and unjust"[9,p.5]. On the other hand, the presence of equity can be detected when those who are marginalized in society have access to the highest attainable standard of health, as measured by the health status of the most advantaged.[10] In BC and elsewhere, Indigenous peoples share a disproportionate burden of inequities. There is no universally recognized formal definition of 'Indigenous' peoples, as each community, nation and collectivity has the right to define and identify itself. Indigenous peoples, communities and nations can be generally understood as those that have "a historical continuity with pre-invasion and pre-colonial societies that developed on their territories, [and] consider themselves distinct from other sectors of the societies now prevailing on those territories, or parts of them."[2]

Many researchers have explored the health status of Indigenous populations in Canada and around the world, and further linked this issue to inequities within determinants of health that extend beyond personal behaviour and genetics to encompass broader socio-political factors that influence health in profound ways.[11-16] There is a recent movement in Indigenous health research away from the pathologizing lens that sensationalizes disparities or 'deficiencies' experienced within Indigenous communities, and realigns the focus toward

the role of structural injustices in shaping social conditions.

ELPH does not intend to reproduce findings from other studies that highlight inequitable health status, rather, the purpose of this research is to examine the role of public health systems and structures in the perpetuation or interruption of these inequities. Health care systems, including the public health system, have a mandate to provide services that promote, restore, or maintain the population's health. BC's Guiding Framework for Public Health[17] outlines public health core functions, including: preventing disease, illness and injury; protecting populations from health risks; and promoting healthy public policies, environments and behaviours. Public health has two overarching moral aims: promoting the health of the population and reducing health inequities.[18] The public health system has therefore been identified as an important site for action to promote health equity as well as ameliorate health inequities. However, as a colonial system, it also has the potential to contribute to increased inequities, particularly in relation to existing barriers to accessing health care for Indigenous peoples.

Historical and institutional background

Barriers to health equity for Indigenous peoples can often be manifested in the public health system in the form of fragmented governance, jurisdictional complexity, gaps in service coverage, and lack of government accountability. These issues have been noted and problematized in landmark reports, including the Final Report of the Truth and Reconciliation Commission of Canada,[16] the Final Report of the Royal Commission on Aboriginal Peoples[19] and the Report of the Chief Public Health Officer.[13] Canada's system of Indigenous health governance has been characterized as a "bureaucratic maze"[11,p.5] and "a complex patchwork of policies, legislation and relationships".[20,p.1] These issues can be traced back to the 1867 British North America (BNA) Act, which stipulates that "Indians and the lands reserved for Indians" are a federal jurisdiction (Section 91[24]) and health care, social services and education, are provincial jurisdictions.[21]

The jurisdictional boundaries outlined by the BNA Act may be clear in theory, but have proven to be ambiguous and convoluted in practice. The divisions not only exist across tiers of government, but also translate to divisions across ancestry, places of residence, and land claim agreements. These jurisdictional divisions create confusion over the provision of health services to Indigenous people, and produce overlapping responsibilities among governing authorities at the federal and provincial/territorial levels. Over the years, Indigenous peoples have sought increased control over decisions relating to health policies, programs and services; however, it is unclear whether this has ameliorated or exacerbated jurisdictional complexity.[20,22,24]

British Columbia's public health system

The BC Core Public Health Functions Framework[25] and the subsequent Guiding Framework for Public Health[17] point out that the core functions of public health are the responsibility of the health system at large, non-governmental and private organizations, and civil society; i.e. public health functions are not solely carried out by the formal public health system and traditional public health practitioners. In the ELPH program of research, however, our focus is on the formal public health system in which the aim of policy and practice is health promotion, disease and injury prevention, health protection, and surveillance and assessment, rather than treatment and cure as it is in the larger health care system.

BC's formal public health system is made up of the Ministry of Health, the Provincial Health Services Authority (PHSA), five regional health authorities (Northern Health, Interior Health, Fraser Health, Vancouver Coastal Health, and Island Health), and First Nations Health Authority (FNHA).[26] In December 2001, the provincial government merged the previous 52 health authorities into five in an attempt to streamline a complicated and expensive health care system.[26] The regional health authorities are responsible for planning, managing, and delivering health programs and services within their geographic areas.[26] The Ministry of Health supports and funds the programs and services of all health authorities and provides guidance to ensure a standardized level of quality across geographic regions and populations.[26] The Provincial Health Services Authority also works with the regional health authorities and supporting organizations to plan and coordinate provincial programs and specialized health services throughout the province.[26] These governing authorities work together to provide comprehensive health services to all British Columbians.

FNHA is the newest health authority in BC that specifically represents and serves First Nations people. It is part of a unique health governance framework among First Nations, the Province of BC, and the Government of Canada that is a first of its kind in Canada. A tripartite framework facilitates the transfer of responsibilities for the planning, design, management and delivery of First Nations health programs and services in BC from Health Canada's First Nations and Inuit Health Branch to FNHA. The transfer officially took place on October 1st, 2013, but is the product of extensive consultations and negotiations formalized in a series of three health agreements: the Transformative Change Accord: First Nations Health Plan (2006), the Tripartite First Nations Health Plan (2007), and the BC Tripartite Framework Agreement on First Nation Health Governance (2011).[27] These three agreements form a legally binding framework that outlines the First Nations health governance structure and mandate, the federal and provincial funding commitments, and the unified vision for an integrated health system.

The creation of FNHA does not add to jurisdictional complexity through separate First Nations and non-First Nations health systems; rather, it promotes stronger linkages between FNHA, Health Canada, the BC Ministry of Health, and BC health authorities.[27] Ultimately, coordination and collaboration among these partners should improve the quality, accessibility, effectiveness, and efficiency of health programs and services for First Nations by reducing complexity and promoting more integrated service delivery. The new framework also increases First Nations control over health governance, which enhances the acceptability of these services through incorporation of culturally relevant models of wellness. FNHA has a "community-driven, nation-based" mandate that represents the diversity of BC First Nations peoples and cultures.[22] However, FNHA's mandate applies exclusively to registered First Nations people, and does not address the needs of other Indigenous groups (e.g. Métis, Inuit, non-status) in BC.[20] The federal government distinguishes between registered (or status)

and non-registered (or non-status) Indians. A registered Indian is a person registered under the terms of the Indian Act.[20] It is anticipated that as time goes on, FNHA will play a greater role in influencing the planning and delivery of services to the wider Indigenous population in BC and potentially the non-Indigenous population as well.[27]

Methods and results

Development of the analytic framework

In 2014, CL and JC were asked by the principal investigators to develop an analytic framework to guide the analysis of a case study of Indigenous health equity within ELPH Study 1. The World Health Organization (WHO) framework for social determinants of health[28] and critical Indigenous theory[29] informed development of this framework. Combining the concepts of proximal, intermediate and distal determinants with those related to colonial oppression, CL and JC attempted to shape a framework specifically focused on the relational, systemic and structural environments within which Indigenous public health is shaped. This framework is congruent with theoretical perspectives that inform the larger program of ELPH research,[5] such as intersectionality, which focuses on diverse social locations, forces, and power structures that shape human life.[30,31]

Alexandra Kent (AK) applied the framework to the data gathered from one of BC's health authorities. The details of the pilot case study, including the chosen health authority, have not yet been released to knowledge users or the public. The framework was later named Xpey' Relational Environments, after consultation with Shauna Underwood, an Indigenous Advisor at the University of Victoria. Xpey' means western red cedar in Hul'q'umi'num', a dialect within the Halkomelem language group spoken primarily by First Nations on Vancouver Island. There is no one word for tree in Hul'q'umi'num'; therefore, we chose western red cedar for its cultural significance as a sacred medicine.

Xpey' Relational Environments is adapted from a tree metaphor previously developed by CL to represent proximal, intermediate, and distal determinants of Indigenous health.[15] The land-based metaphor uses biomimicry to provide a deeper analysis of abstract concepts through an understanding of the natural world.[32] While not specific to Indigenous cultures, the tree metaphor offers an Indigenous cultural sensibility. CL explained the tree metaphor as follows:

> *We typically think of trees as possessing three interconnected elements: the crown (leaves and branches), the trunk, and the roots. Each part of the tree is dependent not only upon the other parts for sustenance and support, but also upon the environment that nourishes and sometimes damages them.*[33,p.4]

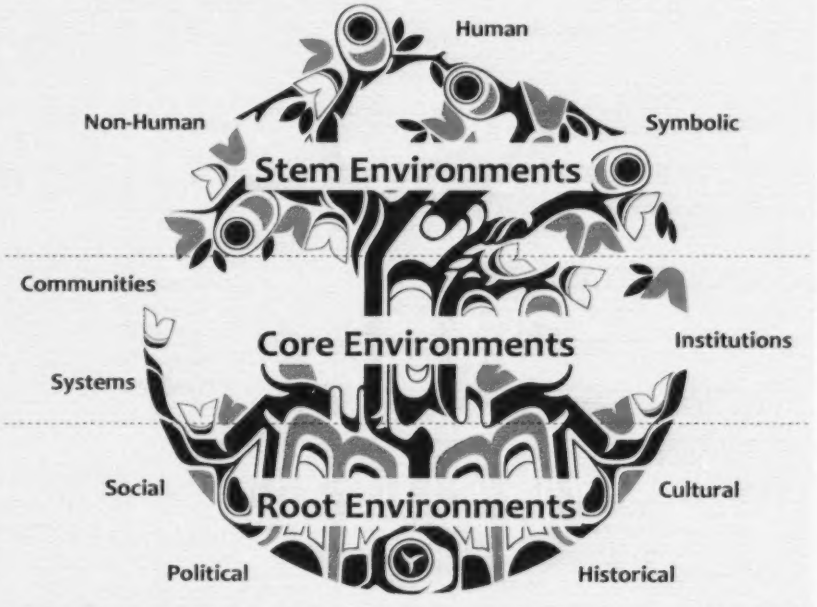
Whereas the health of the unseen roots strongly influences the health of the tree, the condition of the crown is often an indicator of the tree's overall health. Xpey' Relational Environments applies this understanding to the physical and theoretical settings, or 'relational environments', in which health equity is manifested in public health systems and structures.

Within Xpey', relational environments are conceptualized as the three elements of a tree: stem, core, and roots (Figure 1). Like the crown of a tree, stem environments influence individual and community health in the most obvious and direct ways,[33] encompassing interpersonal relationships such as those between service providers and clients or patients; the natural and built environment, including barriers to services and resources; and the symbolic positioning or representation of people's intersectional identities and cultures.

Like the trunk of a tree, core environments connect stem and root environments in ways that can facilitate or hinder health.[33] These relational environments have a less direct influence on the health of individuals, but they strongly influence the relationships and settings within the stem environment. Core environments include: systems of authorities, policies and bureaucracies; leadership and management within relevant institutions and organizations; and the local systems and structures at the community level.

Finally, root environments represent the historical, political, social and cultural contexts from which all other relational environments evolve.[15] CL explained, "Just as maladies observed in the leaves are generally not the cause of unhealthy trees, inequities in human health frequently result from corruption or deficiencies in the unseen but critical root system"[33,p.5]. For Indigenous health and health governance, these roots take the

FIGURE 1
Visual representation of Xpey' Relational Environments framework



Source: Authors obtained permission from artist, kireihiryu, to use tree illustration.

form of colonial histories and intergenerational trauma, political relationships and arrangements, social and material inequities, and cultural connection or loss. Among these stem, core, and root relational environments, there are protective features that promote well-being and resilience, as well as risk factors that can produce unfavorable circumstances or hinder health.

Application of the analytic framework

Over the last four years, the ELPH team has undertaken a review of strategic plans, service plans, health equity plans, government reports, and other relevant documents in each of the health authorities. Document collection has occurred alongside in-depth interviews and focus groups with frontline staff, supervisors, managers and senior executives. Both sources of data were collected for baseline assessment in 2012/2013 and again for follow-up, which started in the summer of 2015 and came to a close in the spring of 2016.

All data from the pilot case study selected for analysis with the Xpey' Relational Environments framework were compiled and coded in NVivo10 qualitative software version 10 (QSR International Pty Ltd. 2012), a qualitative software package that helps store, organize, manage and analyze qualitative data.[34] Initial codes were derived deductively, using Xpey' Relational Environments for higher-level categorization into stem, core, and root environments. Inductive coding was also used to capture the depth and contextual detail of the content. NVivo supported the development of 'in vivo' code labels through use of word frequency queries to identify recurring terms and concepts in the sources. Data-driven codes serve as subcodes within the overarching theory-driven codes (stem, core and root environments). Once a point of saturation was reached, codes were refined and manually sorted by placing categories into relationship with others based on conceptual similarities until the best fit was achieved. As the code hierarchy was integrated into the theoretical framework, the relational environments came to life.

In 2015, CL secured a CIHR Planning and Dissemination Grant to hold a stakeholder engagement meeting with representatives from the BC public health system who are responsible for Indigenous health in their respective organizations. In early 2016, a one-day gathering of leaders from the Indigenous health departments of each of the organizations in BC's public health system was held, including the five regional health authorities, FNHA, the Provincial Health Services Authority, and the Ministry of Health. The majority of those in attendance were First Nations or Métis peoples who work closely with communities across the province. Preliminary findings from the pilot case study were presented to participants to solicit feedback on the Xpey' Relational Environments framework.

Several significant reflections and key recommendations came out of the stakeholder engagement meeting. Everyone in attendance agreed that Indigenous health equity is a critical issue in BC's public health system and that it should be a research priority. The stakeholders were also very supportive of Xpey' Relational Environments as a framework for analyzing Indigenous health equity. However, some participants expressed concern that ELPH was not originally conceived as an Indigenous health equity project, and they pointed out the limitations of adding an Indigenous focus through secondary analysis rather than situating the research in an Indigenous approach from the onset.

Discussion

ELPH is theoretically grounded in intersectionality[30,31] and social justice theory,[18,35] as opposed to post-colonial, critical race theory or Indigenous world views, which is a significant and important limitation restricting use and application of the Xpey' Relational Environments framework within ELPH. ELPH is a collaborative research project with public health leaders within BC's health authorities (which began in 2011 before FNHA was formally introduced to BC's public health system and at a time when Indigenous departments had less presence within health authorities). ELPH research leads met with FNHA representatives once FNHA was established to invite them to be a partner. The research was already well in progress at the time, so we respected their decision not to partner and agreed to share updates and findings throughout the research process. Thus, data collected in ELPH does not reflect important work being done by and with Indigenous peoples within health authorities, in part, due to lack of partnerships and timing of the project. This creates a significant gap in the representation of Indigenous health equity work in BC's public health system.

The initial work and subsequent feedback generated during the stakeholder engagement meeting was invaluable and provided important guidance for the application of the Xpey' Relational Environment framework in future health equity research endeavours. Specifically, the use of the Xpey' Relational Environments framework as an analytic structure must be grounded within Indigenous research methodologies and led by Indigenous peoples. Furthermore, future applications of the framework—or adaptations of it—should include careful representation and consideration of the unique experiences of specific cultural groups (e.g. Métis). With further input into the framework from Indigenous Knowledge Holders and organizational leaders as well as the engagement of Indigenous-led research approaches, the Xpey' Relational Environments framework could have utility in framing future research questions related to Indigenous equity in BC public health and elsewhere.

Conclusion

This application of Xpey' Relational Environments serves as an example of the analytic framework's utility for exploring and conceptualizing Indigenous health equity. The framework captures the critical importance of the determinants of Indigenous health equity in BC's formal public health system, but is also transferable to other studies and other contexts. One obvious application for future development is a focused analysis of FNHA's strategies to reduce inequities and enhance health equity for Indigenous peoples. Furthermore, Xpey' Relational Environments could be used as an analytic framework to explore local health systems within an Indigenous community context, using community-based participatory action research. Through our experience applying Xpey' Relational Environments to a pilot case study within the ELPH research program, we recognize that any future applications of this framework need to be situated within an Indigenous research process, informed by cultural protocols and guided by Elders or Knowledge Holders.

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Conflicts of interest

The authors declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Authors' contributions and statement

BP and MM contributed to the study concept, research design and data collection. CL and JC provided methodological input and designed the analytic framework. AK piloted the framework, interpreted the findings and wrote the manuscript, with guidance from co-authors. All authors assisted in manuscript revision and approved the final version.

The views expressed in this article are the views of the authors and should not be taken to represent the views of the funders.

The content and views expressed in this article are those of the authors and do not necessarily reflect those of the Government of Canada.

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Urinary bisphenol A and obesity in adults: results from the Canadian Health Measures Survey

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Abstract

Introduction: Exposure to bisphenol A (BPA) has been shown to affect lipid metabolism and promote weight gain in animal studies. Recent epidemiological studies also support a link between BPA and obesity in human populations, although many were limited to a single adiposity measure or have not considered potential confounding by dietary factors. The purpose of this study is to examine associations between urinary BPA and adiposity measures in a nationally representative sample of Canadian adults.

Methods: We performed analyses using biomonitoring and directly measured anthropometric data from 4733 adults aged 18 to 79 years in the Canadian Health Measures Survey (2007–2011). We used multinomial and binary logistic regression models to estimate associations of urinary BPA with body mass index (BMI) categories (overweight vs. under/normal weight; obesity vs. under/normal weight) and elevated waist circumference (males: ≥ 102 cm; females: ≥ 88 cm), respectively, while controlling for potential confounders. Linear regression analyses were also performed to assess associations between urinary BPA and continuous BMI and waist circumference measures.

Results: Urinary BPA was positively associated with BMI-defined obesity, with an odds ratio of 1.54 (95% confidence interval [CI]: 1.002–2.37) in the highest (vs. lowest) BPA quartile (test for trend, $p = .041$). Urinary BPA was not associated with elevated waist circumference defined using standard cut-offs. Additionally, each natural-log unit increase in urinary BPA concentration was associated with a 0.33 kg/m^2 (95% CI: 0.10–0.57) increase in BMI and a 1.00 cm (95% CI: 0.34–1.65) increase in waist circumference.

Conclusion: Our study contributes to the growing body of evidence that BPA is positively associated with obesity. Prospective studies with repeated measures are needed to address temporality and improve exposure classification.

Keywords: bisphenol A, endocrine disruptors, obesity, body mass index, waist circumference, biomonitoring, Canadian Health Measures Survey

Highlights

- This is the first Canadian study to investigate the association between bisphenol A (BPA) and adiposity measures.
- A higher level of urinary BPA is associated with greater odds of being obese among Canadian adults 18 to 79 years of age.
- Urinary BPA concentration is also positively associated with continuous measures of adiposity, including body mass index and waist circumference.
- Prospective studies with repeated measures are needed to address temporality and improve exposure classification.

Introduction

Obesity is a growing epidemic worldwide and is expected to exceed smoking as a leading contributor to the burden of chronic disease.[1,2] In Canada, one in four adults is obese.[3] Excess consumption of energy-dense foods, inadequate physical activity, and increased sedentary behaviour have been identified as the most important factors contributing to the obesity epidemic.[4]

Given the dramatic increase in obesity over the past several decades, additional hypotheses are being explored to identify other potentially modifiable risk factors beyond the energy imbalance equation. Ecological studies initially reported a correlation between increasing obesity prevalence and increasing production of synthetic chemicals, including bisphenol A (BPA).[5] BPA is an endocrine-disrupting chemical commonly found in food and beverages stored in polycarbonate plastic and epoxy resin containers.[6] The primary route of exposure to BPA is through dietary intake, although dermal exposure can also occur from skin contact with thermal paper (e.g. receipts, tickets).[6] More than 90% of Canadians[7] and Americans[8] have detectable levels of BPA in their urine, indicating widespread exposure in human populations.

In vitro studies have shown that BPA enhances adipocyte cell differentiation, leading to excess fat accumulation.[9] Rodent studies have also found BPA exposure to increase adipose tissue mass and promote weight gain.[10] More recently,

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evidence of a relationship between BPA and obesity in humans has emerged but is limited to a small number of populations. To date, epidemiological studies in the USA,[11,12] China,[13] and Korea[14,15] have reported positive associations between BPA and adiposity measures in adults. Moreover, as an estrogen-like compound, BPA has been shown to affect males and females differently in animal models,[16] although evidence of sex differences in the association between BPA and obesity in humans is limited.

Since 2007, the Canadian Health Measures Survey (CHMS) has been collecting biomonitoring data and anthropometric measures from a nationally representative sample of Canadians. Given widespread exposure to BPA in Canada and existing evidence supporting the link between BPA and obesity, we evaluated associations between urinary BPA and measures of adiposity in adults aged 18 to 79 years using CHMS data, overall and by sex.

Methods

Data source and study population

This study utilized data from cycles 1 (2007–2009) and 2 (2009–2011) of the CHMS. Details of the CHMS have been described elsewhere.[17,18] Briefly, the CHMS is an ongoing cross-sectional survey that uses a multistage stratified sampling design to collect nationally representative data of the Canadian household population, excluding persons living on reserves or other Aboriginal settlements, full-time members of the Canadian Forces, and residents of institutions and certain remote regions. Cycle 1 collected data from 5604 respondents aged 6 to 79 years at 15 sites across Canada; cycle 2 collected data from 6395 respondents aged 3 to 79 years at 18 sites. The sample for each cycle represented approximately 96% of the Canadian population in the target age range.

The CHMS consists of an in-home household interview capturing sociodemographic, lifestyle, and health characteristics, followed by a mobile examination centre (MEC) visit for direct physical measures, including anthropometric measurements and collection of blood and urine samples. Written informed consent was obtained from each respondent. Protocols were reviewed and approved by Health Canada and the Public Health Agency of Canada (PHAC)'s Research Ethics Board.

Analyses for this study were restricted to adults aged 18 to 79 years at the time of the MEC visit (cycle 1: n = 3726; cycle 2: n = 3873). We pooled data from two CHMS cycles in order to increase sample size and statistical precision of estimates.[19] While all respondents providing urine samples in cycle 1 (n = 3702) were eligible for measurements of BPA, only a random subset of cycle 2 respondents (n = 1117) was selected for these measures, resulting in a combined sample of 4819 respondents. We further excluded pregnant women (n = 37) and respondents with missing urinary BPA and/or creatinine (n = 49), yielding a final sample of 4733 respondents.

Laboratory measurements

Single spot urine samples were collected as midstream urine in cycle 1 and first-catch urine in cycle 2. Cycle 2 respondents were also asked to refrain from urinating 2 hours prior to the MEC visit. The protocol was modified to accommodate new tests for infectious disease markers introduced in cycle 2.[18] Despite these changes, urinary BPA levels were similar for the two cycles.[6] After collection and aliquoting, urine samples were frozen at –20°C and shipped on dry ice to the testing laboratory at the Institut national de santé publique du Québec (Quebec, Canada) for analysis.

Concentrations of total BPA (free and conjugated) were measured using gas chromatography–tandem mass spectrometry based on previously described methods.[7,20] The limit of detection (LOD) was 0.2 µg/L for both cycles. Concentrations below the LOD (n = 436; 9%) were assigned a value of LOD/2 (0.1 µg/L). We categorized urinary BPA concentrations into quartiles (< 0.7 µg/L, 0.7 to 1.2 µg/L, 1.3 to 2.4 µg/L, and > 2.4 µg/L) based on distribution in the overall study population. Additionally, BPA concentrations were natural-log transformed due to skewed distribution and analyzed as a continuous variable.

Urinary creatinine was used to adjust for urine dilution and was measured using the colorimetric end-point Jaffe method.[21] Concentrations below the LOD (cycle 1: 0.035 g/L; cycle 2: 0.050 g/L) were coded as missing and excluded from the analyses (n = 9) as per Statistics Canada's guidelines.[19] To reduce potential bias associated with systematic differences in urinary creatinine concentrations across population characteristics (e.g. sex, age, race/ethnicity), we included creatinine as a covariate in all models instead of standardizing BPA concentrations for creatinine.[22]

Anthropometric measurements

Our primary outcome of interest was body mass index (BMI), derived from height and weight measured using standard procedures.[23] Standing height was measured to the nearest 0.01 cm using a ProScale M150 digital stadiometer (Accurate Technology Inc., Fletcher, NC, USA). Weight was measured to the nearest 0.1 kg using a Mettler Toledo VLC with Panther Plus terminal scale (Mettler Toledo Canada, Mississauga, ON, Canada). BMI, which was calculated as weight (kg) divided by height squared (m^2), was classified into the following categories: underweight (< 18.5 kg/m^2), normal weight (18.5 to 24.9 kg/m^2), overweight (25.0 to 29.9 kg/m^2), obesity class I (30.0 to 34.9 kg/m^2), and obesity class II or III (≥ 35.0 kg/m^2).[4]

As an indicator of abdominal fat, we also examined waist circumference, which was measured to the nearest 0.1 cm at the end of a normal expiration at the mid-point between the bottom of the rib cage and the top of the iliac crest.[4] Central obesity was defined using sex-specific waist circumference cut-offs: ≥ 102 cm in males and ≥ 88 cm in females.[4,24]

Potential confounders

We identified potential confounders according to previous literature examining the BPA–obesity association in adults.[11-15] Sociodemographic variables included sex; age (18 to 29, 30 to 39, 40 to 49, 50 to 59, 60 to 69, or ≥ 70 years); race/ethnicity (white or non-white); highest level of education (less than secondary school degree, secondary school degree, or post-secondary degree); and household income adequacy (low/lower middle, upper middle, or high), categorized based on annual household income and number of people living in the household. We also considered lifestyle factors, including smoking status (never, former, or current); alcohol consumption (0 to 3 times/month, 1 to 6 times/week, or daily); and physical activity, which was assessed based on average daily energy expenditure during leisure-time activities reported over the past 3 months and categorized as active

(≥ 3.0 kcal/kg/day), moderately active (1.5 to 2.9 kcal/kg/day), or inactive (< 1.5 kcal/kg/day).

Given that dietary intake is the primary source of BPA exposure and also a known risk factor for obesity, several dietary measures to control for potential confounding were developed utilizing the semi-quantitative food frequency questionnaire in the CHMS, which asked the respondents to report the number of times (per day, week, month, or year) certain types of foods or drinks were consumed over the past year. A diet quality score was derived using a similar approach as the construction of a Mediterranean diet index.[25] First, we grouped food items into seven components (fruits; vegetables; legumes and nuts; cereals and grains; milk/dairy products; fish and seafood; and red/processed meat) and calculated sex-specific median intakes (times per day) for each. With the exception of red/processed meat, all components were considered beneficial and assigned a value of '1' when intake levels were above the median (red/processed meat intake below the median was assigned '1'). The overall diet quality score was determined by summing up values across all components and was categorized as low (0 to 3), medium (4 to 5), or high (6 to 7). In addition, we also examined consumption frequencies of foods or drinks that potentially contain BPA and/or are known to be "obesogenic" due to their high-energy content. These included sugar-sweetened beverages (e.g. regular/non-diet soft drinks, sport drinks, fruit-flavoured drinks), categorized as < 1 time/week, 1 to 6 times/week, or ≥ 1 time/day; and junk food (e.g. French fries, regular-fat potato/tortilla/corn chips, ice cream), categorized as < 1 time/week, 1 to 4 times/week, or ≥ 5 times/week.

Statistical analysis

To account for the complex sampling design of the CHMS, sampling weights were used in all our analyses.[17,18] Variance estimates were obtained using the bootstrap method. Analyses were performed using SAS version 9.3 (SAS Institute Inc., Cary, NC, USA) and SUDAAN version 11.0.1 (Research Triangle Institute, Research Triangle Park, NC, USA). Statistical significance was evaluated at $p < .05$, and tests were two-sided.

Descriptive analyses were conducted to examine distributions of characteristics in the total population and by BPA quartile. The Rao-Scott modified chi-square test was used to determine whether the distribution of BPA quartiles differed across categories of each characteristic. Geometric mean (GM) urinary BPA concentrations were also calculated, and differences were assessed using the *t*-test with Bonferroni-adjusted *p*-values depending on the number of comparisons.

We used multinomial logistic regression to examine associations between urinary BPA quartiles and BMI, categorized as under/normal weight (< 25.0 kg/m^2; reference category), overweight (25.0 to 29.9 kg/m^2), or obesity (≥ 30.0 kg/m^2). Tests for trend across quartiles were performed by modelling the median of each BPA quartile as a continuous variable. We also examined natural log-transformed BPA concentration as a continuous variable in a separate model. Furthermore, to assess the association between urinary BPA and central obesity (i.e. elevated waist circumference), binary logistic regression was performed. All basic models adjusted for sex, age (continuous years),

TABLE 1
Geometric mean and distribution of urinary bisphenol A concentrations across population characteristics among Canadian adults 18 to 79 years of age (n = 4733), 2007–2011 Canadian Health Measures Survey

Characteristics	N[a]	%[b]	GM (µg/L)	95% CI	Urinary BPA quartile, %[b,c]				*p* value[d]
					1	2	3	4	
All	4733	100	1.17	1.10–1.24	26.3	23.3	25.3	25.1	
Sex									.023
Male	2275	49.8	1.27	1.17–1.38	22.7	23.3	27.3	26.8	
Female	2458	50.2	1.08	0.99–1.17	29.9	23.4	23.3	23.5	
Age (years)									.076
18–29	894	21.6	1.35	1.11–1.65	22.2[E]	24.3	25.2	28.3	
30–39	899	17.4	1.34	1.19–1.50	22.5	22.1	27.4	28.1	
40–49	967	21.1	1.07	0.91–1.27	28.8	21.3	22.2	27.7	
50–59	602	18.8	1.18	0.98–1.44	23.6[E]	25.0	31.1	20.3	
60–69	858	13.8	0.99	0.83–1.17	30.8	26.5	22.7	20.0	
≥ 70	513	7.4	0.94	0.78–1.14	38.2	19.2	19.4	23.2	
Race/ethnicity									.096
White	3981	81.4	1.18	1.10–1.26	26.1	24.7	24.2	25.0	
Non-white	747	18.6[E]	1.14	0.93–1.39	27.0	17.2	30.0	25.7	
Highest level of education									.681
Less than secondary school degree	656	12.2	1.11	0.94–1.32	29.0	26.5	21.2	23.2	
Secondary school degree	1271	28.9	1.14	0.99–1.32	25.3	25.7	25.4	23.6	
Post-secondary degree	2758	59.0	1.19	1.10–1.29	26.2	21.9	25.6	26.4	

Continued on the following page

TABLE 1 (continued)
Geometric mean and distribution of urinary bisphenol A concentrations across population characteristics among Canadian adults 18 to 79 years of age (n = 4733), 2007–2011 Canadian Health Measures Survey

Characteristics	N[a]	%[b]	GM (µg/L)	95% CI	Urinary BPA quartile, %[b,c]				p-value[d]
					1	2	3	4	
Household income adequacy									.534
Low/lower middle	1063	19.2	1.29	1.17–1.43	23.9	21.1	29.1	26.0	
Upper middle	1586	31.0	1.09	0.97–1.23	29.0	23.0	25.1	23.0	
High	2084	49.8	1.18	1.08–1.27	25.5	24.5	23.9	26.1	
Smoking status									.445
Never	2365	51.6	1.11	1.02–1.21	27.9	22.4	24.7	25.0	
Former	1416	27.1	1.15	1.05–1.27	26.6	24.1	26.6	22.7	
Current	952	21.3	1.34	1.14–1.58	22.0	24.7	24.9	28.5	
Alcohol consumption									.126
0–3 times/month	2426	50.3	1.16	1.07–1.26	27.5	21.6	24.6	26.3	
1–6 times/week	1890	41.3	1.21	1.12–1.30	23.7	26.3	25.3	24.7	
Daily	417	8.4	1.04	0.85–1.28	31.6	19.3	28.9	20.2	
Physical activity									.566
Active	1037	21.1	1.27	1.12–1.45	24.0	20.7	26.4	28.9	
Moderately active	1215	24.5	1.17	1.02–1.35	26.4	23.0	26.0	24.6	
Inactive	2481	54.5	1.13	1.04–1.22	27.1	24.5	24.5	23.9	
Diet quality score									.515
Low (0–3)	1998	43.7	1.26	1.14–1.39	25.0	21.5	26.4	27.1	
Medium (4–5)	1922	40.1	1.14	1.01–1.27	25.7	24.5	26.2	23.6	
High (6–7)	781	16.2	1.05	0.88–1.24	30.8	24.8	20.8[e]	23.6	
Sugar-sweetened beverage consumption									.182
< 1 time/week	2565	53.2	1.09	1.00–1.20	28.5	24.1	24.8	22.6	
1–6 times/week	1419	31.1	1.26	1.16–1.38	22.9	23.3	26.0	27.8	
≥ 1 time/day	748	15.8	1.26	1.09–1.45	25.5	20.8	25.4	28.4	
Junk food consumption									.095
< 1 time/week	1181	24.6	1.00	0.83–1.20	32.3	25.2	21.3	21.2	
1–4 times/week	2739	57.5	1.21	1.13–1.31	25.0	22.4	26.5	26.1	
≥ 5 times/week	811	17.9	1.29	1.11–1.51	22.0	23.7	27.0	27.3	
Body mass index (kg/m²)									.088
Underweight (< 18.5)	69	1.9[e]	0.84	0.42–1.67	—[f]	49.0[e]	—[f]	—[f]	
Normal weight (18.5–24.9)	1730	37.5	1.09	0.96–1.23	28.6	24.0	24.9	22.6	
Overweight (25.0–29.9)	1737	35.2	1.21	1.08–1.36	24.9	22.2	26.7	26.2	
Obesity class I (30.0–34.9)	744	16.1	1.23	1.00–1.51	28.2	21.6	20.5	29.7	
Obesity class II or III (≥ 35.0)	442	9.3	1.34	1.13–1.59	18.8	22.8[e]	33.0	25.4[e]	
Elevated waist circumference									.823
No	2931	64.7	1.17	1.08–1.27	26.0	22.6	26.4	25.0	
Yes (males: ≥ 102 cm; females: ≥ 88 cm)	1781	35.3	1.17	1.04–1.33	27.0	23.7	23.6	25.7	

Abbreviations: BPA, bisphenol A; CI, confidence interval; GM, geometric mean.

Note: Percentages and GMs were weighted using sampling weights.

[a] Numbers may not sum up to the total (n = 4733) due to missing data for some variables.

[b] Percentages may not sum up to 100% due to rounding.

[c] Quartile 1: < 0.7 µg/L; quartile 2: 0.7–1.2 µg/L; quartile 3: 1.3–2.4 µg/L; quartile 4: > 2.4 µg/L.

[d] *p*-value from the Rao-Scott modified chi-square test comparing the distribution of BPA quartiles across categories of each characteristic.

[e] Interpret with caution (coefficient of variation is between 16.6% and 33.3%).

[f] Data do not meet Statistics Canada's guidelines for release due to extreme variability (coefficient of variation > 33.3%).

and urinary creatinine concentration (continuous g/L). We then constructed multivariate models by adding individual variables from the list of potential confounders to the basic models. Variables were included in the final models if they were associated with both the exposure (BPA quartile; $p < .20$ from chi-square test) and the outcome (BMI or waist circumference category; entered the regression model at $p < .20$), or if they changed the sex-, age-, and creatinine-adjusted odds ratio (OR) for BPA by > 10%. In addition to sex, age, and urinary creatinine, all BMI and waist circumference models adjusted for race/ethnicity, alcohol consumption, and junk food consumption; waist circumference models additionally adjusted for sugar-sweetened beverage consumption. We also estimated associations of urinary BPA (as quartiles or natural log-transformed continuous variable) with continuous measures of BMI and waist circumference using linear regression. Additionally, we stratified our analyses to explore potential effect modification by sex. Statistical significance of multiplicative interaction terms (sex × BPA) was also tested.

We performed a few sensitivity analyses. First, since fasting status (≥ 10 or < 10 hours) and time of urine collection (morning, afternoon, or evening) may be associated with urinary BPA levels,[26,27] we assessed potential confounding by these variables. Second, we restricted our models to respondents aged 18 to 64 years, as standard BMI and waist circumference classifications may not be applicable to elderly adults.[24] Third, we re-ran the models excluding respondents with self-reported health professional-diagnosed chronic conditions (cardiovascular disease [including heart attack, stroke, and any heart disease], diabetes, and/or kidney disease) that may be related to obesity and BPA exposure/excretion.[28,29] Fourth, since the distribution of full-sample urinary BPA quartiles differed between males and females, we repeated sex-stratified models using sex-specific instead of full-sample quartiles.

Results

Characteristics of the study population, including urinary BPA concentrations, are presented in Table 1. Overall, the GM urinary BPA concentration was 1.17 µg/L

TABLE 2
Associations between urinary bisphenol A and overweight and obesity (vs. under/normal weight) in adults 18 to 79 years of age, overall and by sex, 2007–2011 Canadian Health Measures Survey

Urinary BPA concentration	Model 1[a]					Model 2[b]				
	N	Overweight		Obesity		N	Overweight		Obesity	
		OR	95% CI	OR	95% CI		OR	95% CI	OR	95% CI
Overall										
Quartile 1 (< 0.7 µg/L)	1345	1.00	Ref	1.00	Ref	1342	1.00	Ref	1.00	Ref
Quartile 2 (0.7–1.2 µg/L)	1010	0.94	0.59–1.50	1.05	0.71–1.54	1009	0.91	0.58–1.44	1.01	0.69–1.48
Quartile 3 (1.3–2.4 µg/L)	1210	1.10	0.73–1.67	1.23	0.83–1.81	1208	1.09	0.70–1.70	1.26	0.85–1.87
Quartile 4 (> 2.4 µg/L)	1157	1.18	0.76–1.83	1.56	1.02–2.38	1156	1.14	0.73–1.77	1.54	1.002–2.37
p-trend[c]			.322		.036			.394		.041
Log BPA (continuous)	4722	1.05	0.90–1.22	1.15	1.01–1.31	4715	1.03	0.89–1.20	1.15	1.004–1.31
Males										
Quartile 1 (< 0.7 µg/L)	537	1.00	Ref	1.00	Ref	537	1.00	Ref	1.00	Ref
Quartile 2 (0.7–1.2 µg/L)	483	1.14	0.57–2.27	1.26	0.64–2.49	483	1.05	0.52–1.14	1.08	0.52–2.25
Quartile 3 (1.3–2.4 µg/L)	635	1.44	0.72–2.90	1.42	0.64–3.17	633	1.41	0.69–2.92	1.45	0.60–3.49
Quartile 4 (> 2.4 µg/L)	618	1.51	0.81–2.82	1.77	0.78–4.01	618	1.43	0.74–2.74	1.60	0.67–3.80
p-trend[c]			.208		.193			.248		.266
Log BPA (continuous)	2273	1.17	0.95–1.45	1.19	0.94–1.52	2271	1.16	0.93–1.44	1.17	0.91–1.51
Females										
Quartile 1 (< 0.7 µg/L)	808	1.00	Ref	1.00	Ref	805	1.00	Ref	1.00	Ref
Quartile 2 (0.7–1.2 µg/L)	527	0.83	0.42–1.65	0.90	0.49–1.66	526	0.84	0.42–1.66	0.93	0.52–1.68
Quartile 3 (1.3–2.4 µg/L)	575	0.86	0.45–1.64	1.12	0.56–2.23	575	0.86	0.44–1.67	1.16	0.60–2.27
Quartile 4 (> 2.4 µg/L)	539	0.95	0.49–1.85	1.41	0.72–2.75	538	0.93	0.46–1.86	1.47	0.77–2.81
p-trend[c]			.960		.211			.973		.169
Log BPA (continuous)	2449	0.92	0.77–1.11	1.12	0.92–1.36	2444	0.91	0.75–1.10	1.12	0.93–1.36

Abbreviations: BPA, bisphenol A; CI, confidence interval; OR, odds ratio; Ref, reference category.

[a] Adjusted for sex (overall model only), age, and urinary creatinine concentration.

[b] Adjusted for Model 1 covariates plus race/ethnicity, alcohol consumption, and junk food consumption.

[c] *p*-value for test of trend calculated by modelling the median of each BPA quartile as a continuous variable.

(95% confidence interval [CI]: 1.10–1.24). GMs decreased with age and were significantly higher among males and those who consumed junk food ≥ 5 times/week (all $p < .05$; data not shown). With the exception of sex ($p = .023$), the distribution of BPA quartiles did not differ by any of the characteristics.

As shown in Table 1, 60% of Canadian adults were overweight (35%) or obese (25%), and 35% had an elevated waist circumference (i.e. centrally obese). GM urinary BPA concentrations increased with BMI, from 0.84 µg/L (95% CI: 0.42–1.67) and 1.09 µg/L (95% CI: 0.96–1.23) among underweight and normal weight individuals, respectively, to 1.34 µg/L (95% CI: 1.13–1.59) among those in the class II/III obese categories (p-trend = 0.06). BPA concentrations did not differ by waist circumference category.

Table 2 presents associations of urinary BPA with BMI-defined overweight and obesity, overall and by sex. In the overall model adjusted for sex, age, and urinary creatinine (model 1), respondents in the highest BPA quartile had a significantly higher odds of being obese (vs. under/normal weight) compared to those in the lowest quartile (OR = 1.56, 95% CI: 1.02–2.38), with an increasing trend across increasing quartiles (p-trend = .036). Results remained largely unchanged following additional adjustment for potential confounders (model 2) (OR [quartile 4 vs. 1] = 1.54, 95% CI: 1.002–2.37; p-trend = .041). Similarly, natural log-transformed BPA (continuous) was positively associated with obesity (OR = 1.15, 95% CI: 1.004–1.31). For the overweight category, associations were generally positive but nonsignificant, with an OR of 1.14 (95% CI: 0.73–1.77) in the highest (vs. lowest) BPA quartile. When results were examined by sex, associations between urinary BPA and both overweight and obesity did not reach statistical significance in either sex. Although there was no evidence of a significant sex × BPA interaction ($p > .05$; data not shown), we observed stronger positive associations in males than females for both overweight (OR [quartile 4 vs. 1] = 1.43 vs. 0.93) and obesity (OR [quartile 4 vs. 1] = 1.60 vs. 1.47).

We did not find significant associations between urinary BPA and central obesity, overall (OR [quartile 4 vs. 1] = 1.16, 95% CI: 0.81–1.66; p-trend = .463) or by sex (Table 3). Sex-stratified models suggested a stronger association in males than females (OR [quartile 4 vs. 1] = 1.28 vs. 1.03), although a statistically significant interaction was not found ($p > .05$; data not shown). Additional adjustment for height in the models to control for overall stature did not alter the results (data not shown).

Figure 1 presents associations of urinary BPA quartile with continuous measures of BMI and waist circumference. Respondents in the third and fourth BPA quartiles had significantly greater BMI (1.03 kg/m^2, 95% CI: 0.30–1.76; and 1.06 kg/m^2, 95% CI: 0.18–1.93, respectively) compared to those in the first quartile. A similar pattern was observed for waist circumference, although statistical significance was reached in the third (2.42 cm, 95% CI: 0.46–4.39) but not the fourth (2.73 cm, 95% CI: –0.14 to 5.60) quartile. Furthermore, each natural-log increase in urinary BPA concentration was associated with a 0.33 kg/m^2 (95% CI: 0.10–0.57) increase in BMI and a 1.00 cm (95% CI: 0.34–1.65) increase in waist circumference (Table 4), with slightly stronger associations in females (p for interaction > .05; data not shown).

In our sensitivity analyses (data available by request), inclusion of fasting status or time of urine collection did not change effect estimates by > 10% in any of the models, indicating that these variables did not confound the associations observed. Next, analyses restricted to adults aged 18 to 64 years yielded similar results, with

TABLE 3
Associations between urinary bisphenol A and central obesity[a] in adults 18 to 79 years of age, overall and by sex, 2007–2011 Canadian Health Measures Survey

Urinary BPA concentration	Model 1[b]			Model 2[c]		
	N	OR	95% CI	N	OR	95% CI
Overall						
Quartile 1 (< 0.7 µg/L)	1342	1.00	Ref	1339	1.00	Ref
Quartile 2 (0.7–1.2 µg/L)	1003	1.12	0.76–1.67	1001	1.12	0.75–1.65
Quartile 3 (1.3–2.4 µg/L)	1211	0.96	0.64–1.45	1209	0.99	0.67–1.46
Quartile 4 (> 2.4 µg/L)	1156	1.16	0.81–1.65	1155	1.16	0.81–1.66
p-trend[d]			.470			.463
Log BPA (continuous)	4712	1.06	0.96–1.16	4704	1.05	0.96–1.16
Males						
Quartile 1 (< 0.7 µg/L)	537	1.00	Ref	537	1.00	Ref
Quartile 2 (0.7–1.2 µg/L)	479	1.41	0.88–2.26	478	1.27	0.80–2.02
Quartile 3 (1.3–2.4 µg/L)	636	1.31	0.86–1.99	634	1.32	0.89–1.98
Quartile 4 (> 2.4 µg/L)	618	1.41	0.78–2.55	618	1.28	0.72– 2.27
p-trend[d]			.434			.616
Log BPA (continuous)	2270	1.08	0.92–1.28	2267	1.07	0.91–1.26
Females						
Quartile 1 (< 0.7 µg/L)	805	1.00	Ref	802	1.00	Ref
Quartile 2 (0.7–1.2 µg/L)	524	0.95	0.55–1.65	523	1.01	0.57–1.77
Quartile 3 (1.3–2.4 µg/L)	575	0.76	0.43–1.36	575	0.80	0.45–1.44
Quartile 4 (> 2.4 µg/L)	538	1.00	0.60–1.66	537	1.03	0.64–1.65
p-trend[d]			.902			.864
Log BPA (continuous)	2442	1.03	0.87–1.18	2437	1.03	0.90–1.18

Abbreviations: BPA, bisphenol A; CI, confidence interval; OR, odds ratio; Ref, reference category.

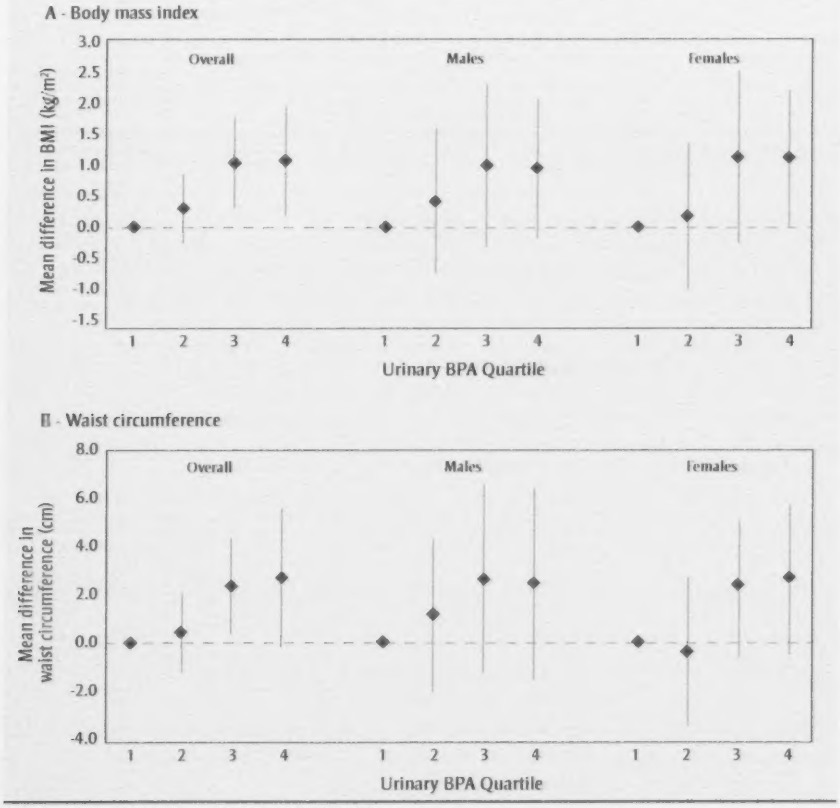
[a] Waist circumference ≥ 102 cm in males and ≥ 88 cm in females.

[b] Adjusted for sex (overall model only), age, and urinary creatinine concentration.

[c] Adjusted for Model 1 covariates plus race/ethnicity, alcohol consumption, junk food consumption, and sugar-sweetened beverage consumption.

[d] p-value for test of trend calculated by modelling the median of each BPA quartile as a continuous variable.

FIGURE 1
Mean difference in (A) body mass index and (B) waist circumference across urinary bisphenol A quartiles (vs. quartile 1) in adults 18 to 79 years of age, overall and by sex, 2007–2011 Canadian Health Measures Survey



Abbreviations: BMI, body mass index; BPA, bisphenol A.

Notes: Quartile 1: < 0.7 µg/L; quartile 2: 0.7–1.2 µg/L; quartile 3: 1.3–2.4 µg/L; quartile 4: > 2.4 µg/L.

All models adjusted for sex (overall models only), age, urinary creatinine concentration, race/ethnicity, alcohol consumption, and junk food consumption. Models for waist circumference additionally adjusted for sugar-sweetened beverage consumption. Error bars represent 95% confidence intervals.

slightly stronger associations for obesity (OR [quartile 4 vs. 1] = 1.64, 95% CI: 1.01–2.66; *p*-trend = .018), as compared to analyses conducted on the full sample. Similarly, when we restricted our analyses to respondents without specific chronic conditions, results did not change appreciably. Finally, when sex-stratified analyses were repeated using sex-specific instead of full-sample quartiles, effect estimates changed slightly (e.g. OR for obesity was attenuated in both sexes for quartile 4 vs .1) but were generally in the same direction, with stronger positive associations consistently observed in males.

Discussion

Using data from the CHMS, our study provides, for the first time, an examination of associations between BPA and indicators of obesity in the general Canadian adult population. We found that urinary BPA was positively associated with BMI-defined general obesity in a dose-dependent manner and that this association persisted after controlling for potential confounders, including diet-related factors. These findings are consistent with other large-scale cross-sectional studies of adults in the USA[11,12] and China,[13] as well as with studies in children.[30-33] Effect sizes in our study were slightly lower than those reported using the US National Health and Nutrition Examination Survey[11] (OR [quartile 4 vs. 1] = 1.76) and were more similar with the study in Chinese adults[13] (OR [quartile 4 vs. 1] = 1.50). A possible explanation for the weaker associations, compared to US results, may be related to the substantially lower BPA concentrations in our study and the Chinese study. In addition, while our results demonstrated increasing odds of obesity across increasing urinary BPA quartiles, some studies reported a potential threshold or non-monotonic effect.[11,15,32] Although non-monotonic associations have been commonly observed for BPA and other endocrine disruptors in toxicological studies,[34] ubiquitous low-level exposures to BPA in human populations present a challenge for assessing and interpreting dose-response relationships.[35]

Consistent with previous studies,[11,13,14] our study also provides evidence for positive associations between urinary BPA and continuous BMI and waist circumference measures. However, unlike studies in the USA[11,12] and Asia,[13,14] we did not find an association between urinary BPA and central obesity defined using waist circumference cut-offs. Although we used the same cut-offs as the US studies, differences in racial/ethnic makeup between the populations may have contributed to the discrepant findings.[36] Furthermore, it has been shown that urinary BPA concentration differed by race/ethnicity and was significantly higher among black compared to white Americans.[27] However, owing to small sample sizes of individual ethnic groups within our study population, we could not further control for race/ethnicity or examine race/ethnicity-specific associations.

Several biological mechanisms have been proposed to explain the association between BPA and obesity, although the exact mode of action remains unclear. For example, BPA has been shown to promote adipocyte differentiation and fat accumulation,[9] as well as bind to estrogen receptors on adipocytes and inhibit the release of the hormone adiponectin.[37] While most animal studies focused on in utero exposure, Miyawaki et al.[10] showed that BPA exposure during both perinatal and postnatal periods led to weight gain in mice. Additionally, long-term exposure to BPA in adult mice was shown to increase adipose tissue mass and induce insulin resistance, hyperglycemia, and hypercholesterolemia,[38] suggesting the role of BPA in the development of obesity and cardiometabolic dysfunction. Similarly, epidemiological studies provide growing evidence that, in addition to obesity, exposure to BPA may be associated with increased risk of diabetes and cardiovascular disorders.[39]

TABLE 4
Associations between log-transformed urinary bisphenol A and continuous measures of body mass index and waist circumference in adults 18 to 79 years of age, overall and by sex, 2007–2011 Canadian Health Measures Survey

	Body mass index (kg/m²)[a]			Waist circumference (cm)[b]		
	N	β[c]	95% CI	N	β[c]	95% CI
Overall						
Log BPA (continuous)	4715	0.33	0.10 to 0.57	4704	1.00	0.34 to 1.65
Males						
Log BPA (continuous)	2271	0.28	–0.04 to 0.59	2267	0.72	–0.22 to 1.67
Females						
Log BPA (continuous)	2444	0.38	0.73 to 2.28	2437	1.16	0.39 to 1.92

Abbreviations: BPA, bisphenol A; CI, confidence interval.

[a] Adjusted for sex (overall model only), age, urinary creatinine concentration, race/ethnicity, alcohol consumption, and junk food consumption.

[b] Adjusted for sex (overall model only), age, urinary creatinine concentration, race/ethnicity, alcohol consumption, junk food consumption, and sugar-sweetened beverage consumption.

[c] Mean change in body mass index (kg/m²) or waist circumference (cm) per natural-log unit increase in urinary BPA concentration.

Like most epidemiological studies, we did not find evidence of effect modification by sex in the association between urinary BPA and obesity. Sex differences in the BPA–obesity association have been implicated in animal studies,[16,40] possibly relating to differences in BPA metabolism[41] and estrogen receptor expression,[42] as well as sex-specific effects of BPA on dietary intake and energy expenditure.[43] Given the biological plausibility, more research is needed to delineate potential sex differences in BPA-induced health outcomes.

Strengths and limitations

This is the first Canadian study to examine associations between BPA and indicators of obesity in adults. Strengths of our study include the population-based design, large sample size, direct anthropometric measurements, high-quality urinary BPA assays, and assessment of multiple potential confounders, including several dietary factors. This is important as recent systematic reviews have pointed out the lack of adjustment for diet, especially processed food consumption, as a major limitation of studies investigating associations between BPA and health outcomes.[26,35,39]

This study has several limitations. First, due to the cross-sectional nature of CHMS data, temporal relationships could not be established. It is possible that obese individuals store, metabolize, and/or excrete BPA differently from non-obese individuals, leading to higher BPA levels in their urine.[44,45] Nonetheless, a recent prospective cohort study of women showed that higher urinary BPA concentration at baseline was associated with greater weight gain during a 10-year follow-up,[46] although additional longitudinal studies in both sexes are warranted. Second, single spot urine measures may not be representative of long-term exposure to BPA. Considerable within-person variability has been shown in urinary BPA measured throughout the day and week[47] and over a period of 1 to 3 years,[48] likely relating to variations in dietary intake. However, our sensitivity analyses showed that time of urine collection and fasting status did not confound the associations observed. Moreover, a recent panel study collected repeated measures of urinary BPA and BMI over a 3-year period and found significant positive associations with overweight in elderly adults regardless of whether single or average BPA measures were analyzed.[15]

Conclusion

In a nationally representative sample of Canadian adults, we showed that urinary BPA was positively associated with general obesity, as well as with continuous BMI and waist circumference. While the imbalance between energy intake and energy expenditure remains a major contributor to obesity, health and economic consequences of obesity attributable to BPA exposure should not be ignored.[49] Furthermore, although BPA is not currently prohibited in Canada, except in baby bottles and cosmetic products, it has been recommended to limit BPA exposure from food packaging given potential effects on health outcomes such as obesity.[6] This underscores the need to further explore the role of BPA as a potential environmental obesogen. Future studies should aim to collect prospective data with repeated measures over extended time periods in order to improve exposure classification and address the temporal relationship between BPA and obesity.

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Conflicts of interest

The authors declare no conflicts of interest.

Authors' contributions and statement

MTD and VCC conceptualized the study, conducted the data analyses, interpreted the data, and drafted the manuscript. MAM and MdG contributed to the interpretation of data, provided expertise and guidance in specific areas, and critically reviewed and revised the manuscript.

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Alcohol consumption and low-risk drinking guidelines among adults: a cross-sectional analysis from Alberta's Tomorrow Project

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Abstract

Introduction: Moderate to heavy alcohol consumption is a risk factor for all-cause mortality and cancer incidence. Although cross-sectional data are available through national surveys, data on alcohol consumption in Alberta from a large prospective cohort were not previously available. The goal of these analyses was to characterize the levels of alcohol consumption among adults from the Alberta's Tomorrow Project in the context of cancer prevention guidelines. Furthermore, we conducted analyses to examine the relationships between alcohol consumption and other high-risk or risk-related behaviours.

Methods: Between 2001 and 2009, 31 072 men and women aged 35 to 69 years were enrolled into Alberta's Tomorrow Project, a large provincial cohort study. Data concerning alcohol consumption in the past 12 months were obtained from 26 842 participants who completed self-administered health and lifestyle questionnaires. We conducted cross-sectional analyses on daily alcohol consumption and cancer prevention guidelines for alcohol use in relation to sociodemographic factors. We also examined the combined prevalence of alcohol consumption and tobacco use, obesity and comorbidities.

Results: Approximately 14% of men and 12% of women reported alcohol consumption exceeding recommendations for cancer prevention. Higher alcohol consumption was reported in younger age groups, urban dwellers, those with higher incomes and those who consumed more red meat. Moreover, volume of daily alcohol consumption was positively associated with current tobacco use in both men and women. Overall, men were more likely to fall in the moderate and high-risk behavioural profiles and show higher daily alcohol consumption patterns compared to women.

Conclusion: Despite public health messages concerning the adverse impact of alcohol consumption, a sizeable proportion of Alberta's Tomorrow Project participants consumed alcohol in excess of cancer prevention recommendations. Continued strategies to promote low-risk drinking among those who choose to drink could impact future chronic disease risk in this population.

Keywords: alcohol, cancer, Alberta's Tomorrow Project, cohort, prevention guidelines

Highlights

- Alcohol consumption is a risk factor for a number of chronic diseases and all-cause mortality.
- Levels of alcohol consumption were reported by 31 072 participants (2001–2009) in Alberta's Tomorrow Project cohort; a geographically-based cohort of adults aged 35 to 69 years.
- Fourteen percent of men and 12% of women reported alcohol consumption exceeding recommendations for cancer prevention.
- Elevated levels of alcohol consumption were positively associated with tobacco use and other risk factors for chronic disease.
- Public health messaging should continue to promote minimal intake levels of alcohol or low-risk drinking to reduce the burden of chronic disease in Alberta.

Introduction

Alcohol contributes substantially to various causes of mortality. Estimates suggest that, globally, alcohol is related to 25.8% of deaths due to injuries, 33.4% of deaths due to diabetes and cardiovascular disease, and 12.5% of cancer-related deaths.[1] Regular alcohol consumption is a known risk factor for at least eight specific types of cancer, including oral cavity, esophagus, pharynx, larynx, female breast, stomach, liver and colorectum.[2,3] The International Agency for Research on Cancer (IARC) has declared ethanol (the active metabolite of alcohol consumption) a Group 1 carcinogen to humans[4], and there is sufficient evidence to suggest a dose-risk relationship between alcohol and adverse health outcomes, especially for cancer[5–9], with no evidence of a threshold effect.[2] Moreover, there does not seem to be any appreciable differences for

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beverage type.[2] Recent population attributable risk estimates predict that 4.2% of all incident cancer cases in the province of Alberta were attributable to alcohol consumption in 2012.[10]

In contrast, light-to-moderate alcohol consumption has previously been shown to have cardioprotective effects[11–14] and provide protection against type II diabetes[15,16] and other chronic diseases.[14,17] However, recent evidence has challenged these findings and suggest that there is no safe limit of consumption, especially for cancer.[18–21] Despite the controversy, identifying a safe threshold based on sound methodology which accounts for beverage type, the frequency and volume of consumption and patterns of use for alcohol remains an important research question.[21] Recent reviews on the topic suggest that even light-to-moderate alcohol use may not be protective for chronic disease.[21] This is contradictory to the messaging that currently exists surrounding alcohol consumption guidelines, which promote moderate alcohol consumption in those who choose to drink.[3,22] Although the rates of past-year drinking among Canadians aged 15 years and older has decreased from 79% in 2004 to 76% in 2013, the rates of risky drinking behaviours have increased.[23] For example, Canada's Low-Risk Drinking Guidelines[24] recommend that women consume no more than 10 drinks per week (with no more than two drinks per day) and for men to consume no more than 15 drinks per week (with no more than three drinks per day).[24,25] Despite these guidelines, the proportion of Canadians who exceed low-risk drinking guidelines continues to rise. Compared to 13.0% in 2004[26], 17.6%[27] and 20.0%[28] of those who drank alcohol (age 25 years and over) exceeded low-risk drinking guidelines for long-term health effects (e.g. cancer, epilepsy, pancreatitis, low birthweight, hemorrhagic stroke, dysrythmias, liver cirrhosis and hypertension) in 2012 and 2013, respectively.

Previous estimates of alcohol consumption prevalence in Alberta have come from national surveys on drug and alcohol use.[26,28–34] Although cross-sectional data are available through national surveys, data on alcohol consumption in Alberta from a large prospective cohort were not previously available. The goal of these analyses was to characterize the levels of alcohol consumption among adults from Alberta's Tomorrow Project in the context of cancer prevention guidelines. Additionally, we identified sociodemographic factors associated with alcohol consumption patterns, its combined prevalence with tobacco use and high-risk profiles, and evaluated the proportion of participants exceeding the World Cancer Research Fund/American Institute of Cancer Research (WCRF/AICR) recommendations for alcohol consumption.

Methods

Alberta's Tomorrow Project is a prospective longitudinal cohort study established to examine the association between various lifestyle factors and chronic disease outcomes, and currently includes 55 000 Albertans aged 35 to 69 years. Detailed information on recruitment methods for Alberta's Tomorrow Project have been published previously.[35,36] In brief, Alberta's Tomorrow Project participants were recruited by random digit dialing (RDD) between 2001 and 2009. The RDD process resulted in 63 486 interested individuals from which 48.8% enrolled into the cohort, resulting in 31 072 participants.[36] Participants completed self-administered questionnaires, including the Health and Lifestyle Questionnaire, the Diet History Questionnaire[37], and the Past Year Total Physical Activity Questionnaire.[38,39] These questionnaires captured information about personal and family health history, cancer screening behaviours, diet and alcohol consumption, smoking habits and environmental exposures. These analyses examine only the first phase of recruited participants who completed the Health and Lifestyle Questionnaire and Diet History Questionnaire. Of the 31 072 cohort participants who enrolled between 2001 and 2009, 86% (n = 26 842) completed information on alcohol consumption.

Assessment of alcohol and variables of interest

Information on alcohol consumption was collected from 2001 to 2009 using a cognitive-based food frequency questionnaire (FFQ) developed by the United States National Cancer Institute as a tool for assessing nutrition over the preceding 12 months[40] and has been adapted for use in Canada.[37] The Diet History Questionnaire (DHQ) was analyzed using Diet*Calc, version 1.4.2 (Canadian version) software. The DHQ has been validated across nutrients and food groups including alcohol. Additionally, numerous other well-designed studies have employed FFQs in their assessment of alcohol consumption.[12,41,42] Participants were queried about consumption frequency and volume of beer, wine/wine coolers, and liquors/mixed drinks during the past year. The questionnaire asked separately about cans/bottles of beer (12-ounce), glasses of wine/wine cooler (5-ounce), and drinks of liquor/mixed drinks (1.5-ounce). Each beverage type had ten frequency response categories ranging from never to six or more servings (drinks) per day over the previous year. We estimated the average amount of ethanol consumed per week using the Canadian standard of 13.6 g of ethanol in a standard drink, corresponding to approximately 341 ml of beer, 142 ml of wine, and 43 ml of liquor.[43] It was not possible to garner information on heavy episodic drinking or whether participants typically drank on weekdays or weekends. We evaluated the proportion of participants who adhered to or exceeded the WCRF/AICR alcohol consumption recommendations for cancer prevention.[44] Individuals were classified as those who adhered (≤ 2 drinks/day for men; ≤ 1 drink/day for women) and those who exceeded recommendations (> 2 drinks/day for men; > 1 drink/day for women).

To estimate the association between alcohol consumption patterns and tobacco use, we examined the proportion of men and women who adhered to or exceeded alcohol consumption guidelines across tobacco use groups. Tobacco use was captured from participant responses to self-report questionnaires at baseline. Participants were asked about their current and former tobacco use histories and were categorized as follows: never, former, current occasional and current daily smoker. Body Mass Index (BMI) was derived from participants' self-measured height and weight, and co-morbidity status was obtained from participants' self-reported physician diagnoses from the baseline questionnaire. To assess prevalence of multiple risk factors, we also considered the prevalence of tobacco smoking, body size (overweight or obesity, defined as body mass index [BMI] > 25 kg/m^2) and presence of comorbidity (defined as self-report of a chronic disease including high blood pressure, angina, high cholesterol, heart attack, stroke, diabetes, polyps in the colon, ulcerative colitis, and cirrhosis of the liver). Multiple risk factors were categorized as none (participants met none of the criteria, i.e. were non-smokers, BMI

< 25 kg/m^2 and reported no chronic conditions), low (met any one of the three criteria), moderate (two of three criteria) and high (all three criteria were met). We then examined the proportion of men and women who were within or exceeded low-risk drinking guidelines within these graded risk categories.

Statistical analysis

Descriptive statistics were used to characterize consumption patterns within the cohort; we examined average consumption of alcohol (0, 0.1 to 4.9, 5 to 14.9, 15 to 29.9, 30 to 44.9, ≥ 45 g/day). Means and standard deviations (SD) were estimated for continuous variables, while frequencies and percentages were estimated for categorical variables. A kappa sensitivity analysis was conducted to determine the agreement between the Diet*Calc estimation of alcohol in number of drinks per day compared to grams of ethanol per day (1 drink = 13.6 g of ethanol). Pearson's chi-square tests were used for all comparison analyses. Additionally, multivariable logistic regression models were used to assess associations between sociodemographic characteristics and WCRF drinking recommendations. Missing data represented < 1% for all included variables. Missing values were omitted from analyses. All statistical tests were performed at a 5% level of significance using SAS version 9.2 (SAS Institute, Cary, NC, USA) on a Linux interface.

Results

Alcohol Consumption Patterns

The majority of participants (84%, n = 22627) reported consuming alcohol at some point in the preceding 12 months. Table 1 presents the proportion of Alberta's Tomorrow Project participants in each alcohol consumption category by sex and sociodemographic characteristics. Median (IQR) consumption of alcohol was 2.1 (5.8) g/day for women and 5.9 (14.8) g/day for men. Compared to non-drinkers, men and women who consumed alcohol tended to be younger, consume more servings of red meat, be of European ethnicity, live in an urban setting, work full-time, and have a household income that exceeded $80000 annually. A clear positive association was observed between daily consumption of alcohol and current tobacco use for both men and women.

World Cancer Research Fund Drinking Recommendations for Cancer Prevention

Table 2 presents the proportion of men and women that fell within or exceeded World Cancer Research Fund recommendations for personal alcohol consumption across demographic categories based on self-reported alcohol consumption. The majority (87%) of cohort participants who reported consuming alcohol in the past 12 months fell within personal recommendations for alcohol consumption, while 13% of participants consumed alcohol in excess of recommendations. Slightly fewer women exceeded the drinking guidelines compared to men (12.1% vs. 13.6%). A higher proportion of men exceeding the recommendations was observed for those who were more educated, had higher annual household incomes, who were middle aged (45 to 54 age group) and divorced/separated/widowed. Similar to men, women exceeding guidelines had higher household incomes, were employed full-time or retired, and were in the 45 to 54 year old age range.

Associations between WCRF drinking guidelines and sociodemographic characteristics are presented in Table 3. Overall, men and women with higher household incomes had higher odds of exceeding WCRF drinking guidelines. Additionally, participants who had ever smoked (current daily, current occasional and former smokers) had a higher odds of exceeding WCRF drinking guidelines compared to never smokers (p < .0001). This was highest for men who were current daily smokers (OR, 95% CI, 3.61, 3.00 to 4.36) and those who were current occasional smokers (OR, 95% CI, 3.56, 2.63 to 4.82). Similar findings were observed for women who smoked daily (OR, 95% CI, 3.06, 2.62 to 3.59) and occasionally (OR, 95% CI, 3.20, 2.43 to 4.21). Women who were of non-European or mixed ethnicity were less likely to exceed guidelines compared to women of European ethnic background (OR, 95% CI, 0.66, 0.51 to 0.85).

Drinking and other risk behaviour patterns

As shown in Table 4, a higher proportion of non-smokers were observed among those who did not consume alcohol. A positive association was observed between current smoking status and total daily alcohol consumption. Volume of alcohol consumption was associated with multiple risk factor categories for both men and women.

Nearly 31.0% of men and 25.4% of women who exceeded guidelines were also current tobacco users (Table 5). The graded/multiple risk factor analysis revealed that a higher proportion of men exceeded the drinking guidelines and had moderate to high-risk profiles compared to women (56.0% vs. 34.6%). Women who exceeded guidelines showed a slightly lower prevalence of multiple risk factors compared to women who fell within the guidelines (35% vs. 37%).

Discussion

We observed that the majority of cohort participants (84%) consumed alcohol in the previous 12 months, which is slightly higher than that reported in other studies on alcohol use in Alberta (76%)[45] and Canada (77.1%).[46] Most participants who reported consuming alcohol in the past 12 months fell within alcohol consumption recommendations for low-risk drinking put forth by the World Cancer Research Fund/American Institute for Cancer Research (WCRF/AICR). However, it should be noted that the cohort only included adults 35 years and older, which excludes those aged 20 to 34 years, known to be the heaviest drinkers in Canada.[23] Globally, the prevalence of alcohol consumption is rising and remains a public health concern.[1] Excess alcohol consumption is widely recognized as a contributor to adverse health outcomes.[1,5,6,8,9,22,47,48] A recent meta-analysis concluded that approximately 34 000 cancer deaths worldwide could be attributed to "light" drinking (defined as: ≤ 12.5 g ethanol or ≤ 1 drink per day) in 2004.[49] The adverse effects of alcohol consumption on health may be underappreciated compared to that of tobacco use, but it has been suggested that the global burden of disease attributable to alcohol was similar to that attributable to smoking exposure in the year 2000.[8,48] Recent findings do not support an overall protective effect from alcohol consumption.[18,50-52] Flawed study designs have been implicated in earlier findings of "protective effects"[53-58] – however, a great deal of controversy on this topic remains.[51,55,59-62]

A large proportion of participants in this study reported light-moderate drinking (0.1 to 29.9 g of ethanol/day or < 1 to 2 drinks/day), and may be unaware of the potential harm associated with even small

TABLE 1
Characteristics of participants according to reported alcohol consumption patterns (g/day)

Characteristics	Total daily consumption of alcohol (g/day)						*p*-value[c]
	0	0.1–4.9	5–14.9	15–29.9	30–44.9	≥ 45	
Men	n = 1342	n = 3327	n = 2708	n = 1546	n = 433	n = 758	
European ethnicity (%)	69.5	73.8	76.8	78.1	80.1	76.1	< 0.0001
Family history of cancer (%)	51.3	48.6	51.0	53.0	54.3	52.6	0.0247
History of colonoscopy or sigmoidoscopy (%)	21.3	20.7	20.4	22.1	22.9	19.8	0.158
Current daily smoker (%)	12.5	12.4	12.5	15.7	18.7	31.1	< 0.0001
Post secondary completed (%)	52.6	55.2	59.6	59.8	56.4	52.0	< 0.0001
Household income ≥ $80,000 (%)	24.7	33.3	42.5	47.0	45.3	36.4	< 0.0001
Full-time occupational status (%)	68.6	74.3	79.2	75.3	73.9	76.8	< 0.0001
Married/living with a partner (%)	82.3	83.3	85.2	83.2	79.0	80.7	0.0024
Living in an urban area (%)	70.8	77.3	78.6	81.2	79.2	76.7	< 0.0001
Age (years)	52.1 (9.4)	50.6 (9.4)	49.7 (8.8)	50.5 (8.9)	50.5 (8.9)	49.9 (8.7)	< 0.0001
Body mass index	28.4 (4.8)	28.3 (4.7)	28.0 (4.2)	27.7 (4.0)	28.2 (4.0)	27.8 (4.0)	< 0.0001
Recreational physical activity (MET h/week)	22.4 (24.7)	25.0 (26.3)	30.9 (27.7)	31.9 (27.4)	32.3 (30.6)	26.8 (27.9)	< 0.0001
No. of pack-years among ever smokers	34.5 (10.3)	32.2 (10.4)	29.5 (9.6)	29.2 (9.5)	29.7 (9.1)	30.7 (9.0)	< 0.0001
Calorie intake from sources other than alcohol (kcal/day)[a]	2185.1 (1110.6)	2046.7 (878.9)	2076.6 (850.5)	2084.6 (820.2)	2250.8 (951.3)	2495.7 (1059.1)	< 0.0001
Red meat in diet (no. servings/week)	5.7 (5.2)	5.4 (4.2)	5.8 (4.1)	6.1 (4.1)	6.8 (5.2)	7.7 (5.5)	< 0.0001
Healthy Eating Index-Canada, 2005[b]	51.1 (9.6)	50.8 (9.3)	50.9 (8.7)	50.5 (8.3)	50.6 (8.0)	50.3 (7.5)	0.272
Women	n = 2873	n = 8688	n = 3346	n = 1329	n = 201	n = 291	
European ethnicity (%)	72.9	77.8	80.2	80.4	83.1	85.2	< 0.0001
Family history of cancer (%)	55.5	55.2	52.9	54.7	54.7	51.2	0.2011
History of colonoscopy or sigmoidoscopy (%)	28.1	24.4	23.3	25.2	20.9	22.3	0.0002
Current daily smoker (%)	13.0	13.9	13.3	17.5	22.9	36.8	< 0.0001
Post-secondary completed (%)	41.6	47.7	55.0	52.0	44.8	38.1	< 0.0001
Household income ≥ $80,000 (%)	16.9	27.9	38.9	39.1	34.8	34.0	< 0.0001
Full-time occupational status (%)	34.1	45.4	47.5	46.0	52.7	50.5	< 0.0001
Married/living with a partner (%)	74.2	74.9	79.0	79.6	77.6	74.2	< 0.0001
Living in an urban area (%)	67.1	76.2	80.3	81.9	75.6	77.7	< 0.0001
Age (years)	51.9 (9.5)	50.2 (9.3)	49.2 (8.7)	50.7 (9.0)	48.6 (8.4)	49.8 (8.2)	< 0.0001
Body mass index	28.5 (6.9)	27.6 (6.1)	26.2 (5.0)	25.8 (4.7)	25.9 (4.6)	26.8 (5.1)	< 0.0001
Recreational physical activity (MET h/week)	17.9 (20.6)	22.5 (22.9)	27.3 (24.2)	29.2 (25.2)	28.4 (24.6)	20.9 (22.7)	< 0.0001
No. of pack-years among ever smokers	32.3 (10.1)	30.3 (9.7)	28.5 (9.1)	29.6 (9.5)	28.9 (7.4)	30.8 (8.5)	< 0.0001
Calorie intake from sources other than alcohol (kcal/day)[a]	1644.2 (720.8)	1574.9 (634.0)	1579.4 (604.3)	1613.4 (629.7)	1681.2 (584.4)	1782.8 (793.4)	< 0.0001
Red meat in diet (no. servings/week)	3.3 (2.7)	3.4 (2.5)	3.6 (2.5)	3.7 (2.5)	4.6 (3.0)	4.0 (2.6)	< 0.0001

Continued on the following page

TABLE 1 (continued)
Characteristics of participants according to reported alcohol consumption patterns (g/day)

Characteristics	Total daily consumption of alcohol (g/day)						*p*-value[c]
	0	0.1–4.9	5–14.9	15–29.9	30–44.9	≥ 45	
Healthy Eating Index-Canada, 2005[b]	55.3 (10.1)	55.4 (9.7)	55.3 (8.9)	54.4 (8.5)	53.6 (7.4)	52.8 (7.3)	< 0.0001
Postmenopause (%)	45.4	53.7	58.1	51.3	62.2	50.5	< 0.0001
Current hormone therapy use (%)	16.3	14.8	13.8	17.0	10.5	15.1	0.0285
Mammogram in past 3 years (%)	79.9	81.5	82.3	85.7	81.4	74.9	< 0.0001

Abbreviation: MET h/week, metabolic equivalent of task hours per week.

Notes: Mean (SD) was presented for continuous variables. Percentages were presented for categorical variables and as column percentages, i.e. 100% within each alcohol consumption category.

[a] 1 kcal = 4.18 kJ.

[b] Without alcohol intake.

[c] The chi-square test was used for categorical variables, and the one-way analysis of variance was used for continuous variables.

but regular amounts of alcohol. Further investigation into the relationship between low-risk drinking and health outcomes is essential to better characterize the exact risk-benefit threshold for alcohol consumption among different population groups. It is likely that current recommendations are not specific enough to account for inter-individual variation, susceptibility to particular disease, and tolerance thresholds.

As previously highlighted by the Pan American Health Organization and the WCRF, alcohol consumption behaviours differ considerably by sex.[3,47] In the present study, men consumed alcohol more frequently and in greater quantities compared to women. Men were twice as likely to report daily drinking compared to women. This gender difference has been observed in previous population-based studies[3,47] and cross-national studies,[63,64] which found higher prevalence of harmful alcohol consumption profiles among men, especially with respect to total volume consumed and risky patterns of use.[63-65] Similar studies have also found that alcohol-attributable disease burden (i.e. cancer, cirrhosis of the liver, neuropsychiatric disorders, etc.) is five times higher in men than women, with a mortality ratio of 10:1 compared to women.[8] The higher consumption observed in men could be attributable to biopsychosocial factors.[63] Similarly, we observed that men were more likely to engage in both higher rates of alcohol consumption and tobacco smoking, amplifying their risk for adverse health outcomes and disease. Both men and women who exceeded drinking guidelines were more likely to use tobacco and have overall higher risk profiles compared to those who fell within current guidelines.

Preliminary analyses from this study suggests that some chronic conditions and comorbidities may be higher among those who exceed WCRF/AICR drinking recommendations, especially for men. Therefore, healthcare providers and public policy initiatives should work within the framework of risk-reduction to determine which strategies may be most appropriate for particular groups of individuals. Interventions targeted at specific populations who are known to have "at risk" alcohol consumption patterns are needed. Given the overwhelming evidence supporting a dose-risk relationship between alcohol and chronic disease, including cancer, public health messaging should continue to focus on limiting heavy drinking and supporting low-risk drinking for individuals who choose to drink, in addition to targeting individuals who may already have a high-risk profile. Future analyses using Alberta's Tomorrow Project will focus on investigating the association between long-term alcohol consumption patterns and incidence of cancer and other chronic diseases in this cohort.

TABLE 2
Proportion of Alberta's Tomorrow Project participants who fall within or exceed the World Cancer Research Fund/American Institute for Cancer Research alcohol consumption recommendations by sociodemographic characteristics[a]

	Men (n = 10 114)			Women (n = 16 728)		
	Within guidelines[b]	Exceed guidelines[c]	*p*-value[d]	Within guidelines[b]	Exceed guidelines[c]	*p*-value[d]
	n (%)	n (%)		n (%)	n (%)	
Totals	8744 (86.5)	1370 (13.6)		14 708 (87.9)	2020 (12.1)	
Age						
35-44	2648 (30.3)	410 (29.9)		4680 (31.8)	597 (29.6)	
45-54	3073 (35.1)	542 (39.6)		5058 (34.4)	809 (40.1)	
55-64	2231 (25.5)	324 (23.7)	0.0021	3650 (24.8)	459 (22.7)	< 0.0001
65-69	792 (9.1)	94 (6.9)		1320 (9.0)	155 (7.7)	
Missing	0 (0.0)	0 (0.0)		0 (0.0)	0 (0.0)	

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TABLE 2 (continued)
Proportion of Alberta's Tomorrow Project participants who fall within or exceed the World Cancer Research Fund/American Institute for Cancer Research alcohol consumption recommendations by sociodemographic characteristics[a]

	Men (n = 10 114)			Women (n = 16 728)		
	Within guidelines[b]	Exceed guidelines[c]	*p*-value[d]	Within guidelines[b]	Exceed guidelines[c]	*p*-value[d]
	n (%)	n (%)		n (%)	n (%)	
Education[e]						
High school not completed	914 (10.5)	144 (10.5)		1359 (9.2)	151 (7.5)	
High school completed	1259 (14.4)	226 (16.5)		3064 (20.8)	421 (20.8)	
Some post-secondary	1599 (18.3)	253 (18.5)	0.3015	3231 (22.0)	432 (21.4)	0.0731
Post secondary completed	4971 (56.9)	747 (54.5)		7053 (48.0)	1016 (50.3)	
Missing	1 (0.01)	0 (0.0)		1 (0.01)	0 (0.0)	
Household income[f]						
< $30 000	804 (9.2)	88 (6.4)		2373 (16.1)	220 (10.9)	
$30 000–$49 000	2189 (25.0)	298 (21.8)		4265 (29.0)	499 (24.7)	
$50 000–$79 000	2393 (27.4)	404 (29.5)	0.0001	3496 (23.8)	487 (24.1)	< 0.0001
≥ $80,000	3224 (36.9)	564 (41.2)		4131 (28.1)	764 (37.8)	
Missing	134 (1.5)	16 (1.2)		443 (3.0)	50 (2.5)	
Occupational status						
Full-time	6563 (75.1)	1041 (76.0)		6413 (43.6)	965 (47.8)	
Part-time	563 (6.4)	93 (6.8)		3419 (23.3)	437 (21.6)	
Unemployed/homemaker/student	221 (2.5)	38 (2.8)	0.5928	2335 (15.9)	270 (13.4)	0.0005
Retired	1129 (12.9)	155 (11.3)		2019 (13.7)	295 (14.6)	
Other	264 (3.0)	43 (3.1)		514 (3.5)	53 (2.6)	
Missing	4 (0.1)	0 (0.0)		8 (0.1)	0 (0.0)	
Marital status						
Married/living with a partner	7324 (83.8)	1098 (80.2)		11 125 (75.6)	1589 (78.7)	
Single (never married)	562 (6.4)	90 (6.6)	0.0011	817 (5.6)	79 (3.9)	0.0043
Divorced/separated/widowed	857 (9.8)	182 (13.3)		2764 (18.8)	352 (17.4)	
Missing	1 (0.01)	0 (0.0)		2 (0.01)	0 (0.0)	
Smoking status						
Current daily	1135 (13.0)	342 (25.0)		1996 (13.6)	414 (20.5)	
Current occasional	269 (3.1)	82 (6.0)		379 (2.6)	98 (4.9)	
Former	3454 (39.5)	595 (43.4)	< 0.0001	5111 (34.8)	947 (46.9)	< 0.0001
Never	3882 (44.4)	350 (25.6)		7209 (49.0)	560 (27.7)	
Missing	4 (0.1)	1 (0.1)		13 (0.1)	1 (0.1)	
Self-reported ethnicity						
European	6535 (74.7)	1063 (77.6)		11 380 (77.4)	1639 (81.1)	
Non-European/mixed ethnicity	589 (6.7)	73 (5.3)	0.0446	831 (5.7)	73 (3.6)	< 0.0001
Missing	1620 (18.5)	234 (17.1)		2497 (17.0)	308 (15.3)	
Geographic location[g]						
Rural	1975 (22.6)	307 (22.4)		3628 (24.7)	399 (19.8)	
Urban	6769 (77.4)	1063 (77.6)	0.8834	11 080 (75.3)	1621 (80.3)	< 0.0001
Missing	0 (0.0)	0 (0.0)		0 (0.0)	0 (0.0)	

Note: Column percentages have been reported, i.e. 100% within each drinking guideline.

[a] Data presented as count and percentage.

[b] Within Guidelines refers to ≤ 2 drinks per day for men and ≤ 1 drink per day for women.

[c] Exceeding Guidelines refers to > 2 drinks per day for men and > 1 drink per day for women.

[d] Indicates statistically significant difference across sociodemographic categories in exceed and meet guidelines using chi-square tests ($p < 0.001$).

[e] Combined responses to: some technical school/college, completed technical school/college, some university degree completed.

[f] Income data are in response to a question about total household income before tax etc.

[g] Geographic location was determined using postal codes, where the "0" as the middle numerical number indicates rural residence.

TABLE 3
Associations between WCRF alcohol intake guidelines and sociodemographic characteristics among participants in the Alberta's Tomorrow Project Cohort Study

Variables	Men				Women			
	OR	95% CI		*p*-value	OR	95% CI		*p*-value
		Lower	Upper			Lower	Upper	
Age (years)	0.99	0.98	1.01	0.0839	1.01	1.00	1.02	0.0221
Body mass index (kg/m²)	0.99	0.97	1.01	0.1463	0.95	0.94	0.96	< 0.0001
Education								
High school completed	1.07	0.83	1.39	0.6047	1.20	0.95	1.51	0.1268
Some post-secondary	1.03	0.80	1.33	0.8217	1.12	0.89	1.42	0.326
Postsecondary completed	1.04	0.82	1.30	0.7674	1.26	1.01	1.58	0.0414
High school not completed[a]	1.00	Ref			1.00	Ref		
Household income								
$30 000–$49 000	1.57	1.15	2.14	0.0041	1.16	0.95	1.41	0.1401
$50 000–$79 000	2.14	1.56	2.92	< 0.0001	1.41	1.14	1.73	0.0013
≥ $80 000	2.47	1.80	3.39	< 0.0001	1.86	1.51	2.30	< 0.0001
< $30 000[a]	1.00	Ref			1.00	Ref		
Occupational status								
Part-time	1.24	0.95	1.63	0.1165	0.93	0.81	1.07	0.3081
Unemployed/homemaker/student	1.14	0.76	1.70	0.5177	0.82	0.69	0.97	0.0198
Retired	1.13	0.88	1.45	0.3528	1.07	0.88	1.29	0.5142
Other	1.25	0.86	1.82	0.2388	0.75	0.54	1.05	0.096
Full-time[a]	1.00	Ref			1.00	Ref		
Marital status								
Married/living with a partner	0.81	0.61	1.08	0.1568	1.23	0.93	1.63	0.1458
Divorced/separated/widowed	1.25	0.90	1.72	0.1824	1.16	0.86	1.55	0.326
Single (never married)[a]	1.00	Ref			1.00	Ref		
Smoking status								
Current daily	3.61	3.00	4.36	< 0.0001	3.06	2.62	3.59	< 0.0001
Current occasional	3.56	2.63	4.82	< 0.0001	3.20	2.43	4.21	< 0.0001
Former	1.92	1.64	2.25	< 0.0001	2.51	2.22	2.84	< 0.0001
Never[a]	1.00	Ref			1.00	Ref		
Self-reported ethnicity								
Non-European or mixed ethnicity	0.77	0.59	0.99	0.0479	0.66	0.51	0.85	0.0015
European[a]	1.00	Ref			1.00	Ref		
Geographic location								
Rural	0.99	0.84	1.16	0.8756	0.82	0.72	0.93	0.0027
Urban[a]	1.00	Ref			1.00	Ref		

Abbreviations: CI, confidence interval; OR, odds ratio; Ref, reference category; WCRF, World Cancer Research Fund.

[a] Reference category.

TABLE 4
The prevalence of self-reported alcohol consumption patterns and risk-related characteristics in Alberta's tomorrow Project cohort[a]

Risk factors	Total daily consumption of alcohol (g/day)						p-value[d]
	0	0.1–4.9	5–14.9	15–29.9	30–44.9	≥ 45	
	n (%)	n (%)	n (%)	n (%)	n (%)	n (%)	
Men							
Totals	1342 (13.3)	3327 (32.9)	2708 (26.8)	1546 (15.3)	433 (4.3)	758 (7.5)	
Never smoker	612 (45.6)	1609 (48.4)	1183 (43.7)	539 (34.9)	129 (29.8)	160 (21.1)	
Current smoker (daily or occasionally)	189 (14.1)	486 (14.6)	451 (16.7)	312 (20.2)	107 (24.7)	283 (37.3)	< 0.0001
Former smoker	539 (40.2)	1230 (37.0)	1074 (39.7)	695 (45.0)	196 (45.3)	315 (41.6)	
Missing	2 (0.2)	2 (0.1)	0 (0.0)	0 (0.0)	1 (0.2)	0 (0.0)	
BMI < 25 kg/m²	319 (23.8)	789 (23.7)	601 (22.2)	361 (23.4)	84 (19.4)	173 (22.8)	0.3328
BMI ≥ 25 kg/m²	1020 (76.0)	2529 (76.0)	2102 (77.6)	1182 (76.5)	348 (80.4)	582 (76.8)	
Missing	3 (0.2)	9 (0.3)	5 (0.2)	3 (0.2)	1 (0.2)	3 (0.4)	
No comorbidities	657 (49.0)	1707 (51.3)	1495 (55.2)	793 (51.3)	205 (47.3)	381 (50.3)	0.0005
Comorbidities[b]	683 (50.9)	1614 (48.5)	1206 (44.5)	749 (48.5)	227 (52.4)	376 (49.6)	
Missing	2 (0.2)	6 (0.2)	7 (0.3)	4 (0.3)	1 (0.2)	1 (0.1)	
No risk[c]	160 (11.9)	439 (13.2)	353 (13.0)	159 (10.3)	36 (8.3)	43 (5.7)	
Low risk[c]	545 (40.6)	1333 (40.1)	1125 (41.5)	631 (40.8)	152 (35.1)	279 (36.8)	< 0.0001
Moderate risk[c]	564 (42.0)	1369 (41.2)	1056 (39.0)	656 (42.4)	205 (47.3)	346 (45.7)	
High risk[c]	73 (5.4)	186 (5.6)	174 (6.4)	100 (6.5)	40 (9.2)	90 (11.9)	
Women							
Totals	2873 (17.2)	8688 (51.9)	3346 (20.0)	1329 (7.9)	201 (1.2)	291 (1.7)	
Never smoker	1636 (56.9)	4251 (48.9)	1385 (41.4)	413 (22.4)	45 (22.4)	39 (13.4)	
Current smoker (daily or occasionally)	400 (13.9)	1429 (16.5)	582 (17.4)	283 (21.3)	67 (33.3)	126 (43.3)	< 0.0001
Former smoker	832 (29.0)	3001 (34.5)	1378 (41.2)	632 (47.6)	89 (44.3)	126 (43.3)	
Missing	5 (0.2)	7 (0.1)	1 (0.03)	1 (0.1)	0 (0.0)	0 (0.0)	
BMI < 25 kg/m²	988 (34.4)	3331 (38.3)	1593 (47.6)	668 (50.3)	94 (46.8)	113 (38.8)	< 0.0001
BMI ≥ 25 kg/m²	1866 (65.0)	5332 (61.4)	1745 (52.2)	659 (49.6)	106 (52.7)	177 (60.8)	
Missing	19 (0.7)	25 (0.3)	8 (0.2)	2 (0.2)	1 (0.5)	1 (0.3)	
No comorbidities	1484 (51.7)	5049 (58.1)	2161 (64.6)	802 (60.4)	129 (64.2)	168 (57.7)	< 0.0001
Comorbidities[b]	1381 (48.1)	3624 (41.7)	1178 (35.2)	524 (39.4)	71 (35.3)	122 (41.9)	
Missing	8 (0.3)	15 (0.2)	7 (0.2)	3 (0.2)	1 (0.5)	1 (0.3)	
No risk[c]	597 (20.8)	2021 (23.3)	988 (29.5)	370 (27.8)	46 (22.9)	39 (13.4)	
Low risk[c]	1062 (37.0)	3355 (38.6)	1334 (39.9)	521 (39.2)	88 (43.8)	112 (38.5)	< 0.0001
Moderate risk[c]	1057 (36.8)	2906 (33.5)	901 (26.9)	369 (27.8)	45 (22.4)	107 (36.8)	
High risk[c]	157 (5.5)	406 (4.7)	123 (3.7)	69 (5.2)	22 (11.0)	33 (11.3)	

Abbreviation: BMI, body mass index.

Note: Results have been presented as column percentages, ie. 100% within each alcohol consumption category.

[a] Multiple risk was evaluated by assessing the following criteria: current tobacco smoking (occasional or daily), body size (overweight or obese, defined as BMI > 25 kg/m²) and presence of comorbidity.

[b] Comorbidity is defined as self-report of a chronic disease including high blood pressure, angina, high cholesterol, heart attack, stroke, diabetes, polyps in colon, ulcerative colitis and cirrhosis of the liver.

[c] Graded risk categories: no risk (participants met none of the criteria above, i.e. were never smokers, BMI < 25 kg/m² and self-reported no chronic condition), low risk (met any one of the three criteria shown above), moderate risk (met two of three criteria shown above) and high risk (met all three criteria shown above).

[d] The chi-square test was used for categorical variables, and the one-way analysis of variance was used for continuous variables.

TABLE 5
Prevalence of alcohol consumption WCRF drinking guidelines and risk-related characteristics[a] in Alberta's Tomorrow Project cohort

Risk factors	WCRF drinking guidelines[b]				*p*-value[c]
	Within guidelines		Exceed guidelines		
	n (%)		n (%)		
Men					
Totals	8744	(86.5)	1370	(13.6)	
Never smoker	3882	(44.4)	350	(25.6)	
Current smoker (daily or occasionally)	1404	(16.1)	424	(31.0)	< 0.0001
Former smoker	3454	(39.5)	595	(43.4)	
Missing	4	(0.1)	1	(0.1)	
BMI < 25 kg/m²	2025	(23.2)	302	(22.0)	0.3680
BMI ≥ 25 kg/m²	6699	(76.6)	1064	(77.7)	
Missing	20	(0.2)	4	(0.3)	
No comorbidities	4560	(52.2)	678	(49.5)	0.0629
Comorbidities[d]	4165	(47.6)	690	(50.4)	
Missing	19	(0.2)	2	(0.2)	
No risk[e]	1087	(12.4)	103	(7.5)	
Low risk[e]	3566	(40.8)	499	(36.4)	< 0.0001
Moderate risk[e]	3571	(40.8)	625	(45.6)	
High risk[e]	520	(6.0)	143	(10.4)	
Women					
Totals	14 708	(87.9)	2020	(12.1)	
Non-smoker	12 320	(83.8)	1507	(74.6)	
Current smoker (daily or occasionally)	2375	(16.2)	512	(25.4)	< 0.0001
Former smoker	5111	(34.8)	947	(46.9)	
Missing	13	(0.1)	1	(0.1)	
BMI < 25 kg/m²	5814	(39.5)	973	(48.2)	0.0035
BMI ≥ 25 kg/m²	8842	(60.1)	1043	(51.6)	
Missing	52	(0.4)	4	(0.2)	
No comorbidities	8551	(58.1)	1242	(61.5)	< 0.0001
Comorbidities[d]	6128	(41.7)	772	(38.2)	
Missing	29	(0.2)	6	(0.3)	
No risk[e]	3540	(24.1)	521	(25.8)	
Low risk[e]	5673	(38.6)	799	(39.6)	< 0.0001
Moderate risk[e]	4813	(32.7)	572	(28.3)	
High risk[e]	682	(4.6)	128	(6.3)	

Abbreviations: BMI, body mass index; WCRF, World Cancer Research Fund.

Note: Results have been presented as column percentages.

[a] Multiple risk was evaluated by assessing the following criteria: tobacco smoking, body size (overweight or obese, defined as BMI > 25 kg/m²) and presence of comorbidity.

[b] Within guidelines refers to ≤ 2 drinks per day for men and ≤ 1 drink per day for women; exceeding guidelines refers to > 2 drinks per day for men and > 1 drink per day for women.

[c] The chi-square test was used for categorical variables, and the one-way analysis of variance was used for continuous variables.

[d] Comorbidity is defined as self-report of a chronic disease including high blood pressure, angina, high cholesterol, heart attack, stroke, diabetes, polyps in colon, ulcerative colitis and cirrhosis of the liver.

[e] Graded risk categories: no risk (participants met none of the criteria above, i.e. were never smokers, BMI < 25 kg/m² and self-reported no chronic condition), low risk (met any one of the three criteria shown above), moderate risk (met two of three criteria shown above) and high risk (met all three criteria shown above).

Limitations

It is important to acknowledge several limitations of the present study. Alberta's Tomorrow Project cohort does not include young adults (< 35 years), who have been shown to have a higher prevalence of alcohol consumption compared to middle-aged adults.[31,34,66] Therefore, these estimates reflect only the adult population of Alberta between the ages of 35 and 69 years. While Alberta's Tomorrow Project was designed to be geographically representative of the adult population of Alberta, no weighted sampling strategy was used in the cohort design. Additionally, the initial recruitment through RDD methods resulted in a 48.4% response rate. It is unknown how responders differed from non-responders as no data were collected on those who did not enroll. While we believe that these results are largely generalizable to adults in Alberta, the data should not be considered representative of the Alberta population as a whole. The exclusion of Albertans under age 35 years may also account for the lower prevalence of Alberta's Tomorrow Project participants who exceed WCRF drinking recommendations compared to other national surveillance data.[31,34,66] In addition, the results of the current analyses are based on participant responses to self-report surveys. Sensitive questions, such as those related to alcohol intake, can often lead to exposure misclassification due to underestimation and underreporting of true consumption.[3,8] An unpublished analysis of the 2004 Canadian Addiction Survey found that respondents indicated they only drink on average one-third of what would be expected from official alcohol sales.[67] A limitation of the use of the Diet History Questionnaire for the assessment of alcohol consumption is that it does not adequately capture heavy episodic or "binge" drinking habits, which may have led to an underestimation of total alcohol consumption. Numerous other well-designed studies have assessed alcohol consumption in a similar fashion, most notably the Nurses' Health Study[11] and the Health Professionals Follow-up study[12], both large ongoing prospective cohort studies.[42]

Conclusion

Despite the potential for underreporting, 84% of participants in the present study reported consuming alcohol in the past year. Men had a median (IQR) consumption of 5.9 (14.8) g/day of alcohol and

women had a median consumption of 2.1 (5.8) g/day. Approximately 14% of men and 12% of women exceeded cancer prevention alcohol consumption recommendations. Additionally, higher volumes of alcohol consumption were found to be associated with tobacco use and elevated risk behaviour profiles in both men and women (all $p < .0001$). Public health messaging that continues to support minimal intake levels or low-risk drinking is essential in promoting moderation among individuals who choose to drink.

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Conflicts of interest

There were no conflicts of interest declared.

Authors' contributions and statement

D.R.B., P.J.R. and C.M.F. were responsible for the study conception. C.M.F., D.R.B., P.J.R., A.E.P., T.R.H. and A.A. contributed substantially to the study design and interpretation of the data. A.A. completed the analyses. D.R.B and T.R.H. were major contributors in writing the manuscript. All authors read and gave final approval of this version to be published and agreed to be guarantors of the work.

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Other PHAC publications

Researchers from the Public Health Agency of Canada also contribute to work published in other journals. Look for the following articles published in 2017:

Hersi M, **Irvine B**, Gupta P, Gomes J, Birkett N, Krewski D. Risk factors associated with the onset and progression of Alzheimer's disease: a systematic review of the evidence. Neurotoxicology. 2017;61:143-87. doi: 10.1016/j.neuro.2017.03.006.

Huet C, Ford JD, **Edge VL**, et al. Food insecurity and food consumption by season in households with children in an Arctic city: a cross-sectional study. BMC Public Health. 2017;17(1):578. doi: 10.1186/s12889-017-4393-6.

Krewski D, Barakat-Haddad C, Donnan J, [...] **Irvine B**, et al. Determinants of neurological disease: synthesis of systematic reviews. Neurotoxicology. 2017;61:266-89. doi: 10.1016/j.neuro.2017.04.002.